THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—41ST YEAR SYDNEY, SATURDAY, FEBRUARY 13, 1954 No. 7

Table of Contents.

HEART DISEASE IN INDUSTRY.[1]

By F. A. E. LAWES,

Sydney.

SINCE industry is composed of people, the title of this address should be "heart disease among people who work with their hands as well as their brains". This is a difficult and debatable subject; for much has been written and spoken on many different aspects of the matter without there being found an answer to the numerous problems presented.

Almost daily opinions are expressed in our courts of law, the expert witnesses being ranged into two opposing camps, and leaving the decision to be made by the judge in most cases, but in others by a jury of men who for most of the time understand but little of the subject under discussion.

I do not propose to review all the literature, which is very large, but rather to indicate some problems and to discuss them and quote examples of cases in which I have been interested.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 27, 1953.

Direct Trauma of the Heart.

The heart may be damaged by penetrating injuries of the chest. Any portion of the heart or pericardium may be damaged. These cases are well recognized and do not require discussion. Non-penetrating injuries of the chest wall may cause bruising of the heart, even though no damage is visible externally. The work of Barber, Beck, Warburg and others has increased our knowledge of these injuries. Necropsy studies long ago showed that fatal rupture of the heart can follow a severe injury to the chest with a blunt instrument; but only recently has it been known that less severe cardiac injuries may occur, with a symptomatic recovery or survival and a variety of persistent and serious cardiac symptoms. Beck believed that there was a failure to recognize, or a fear to diagnose, clinical heart disease due to cardiac contusion. But, as Friedberg remarks, we must be careful "not to attribute to a preceding trauma a host of unrelated cardiac disturbances which are the consequence of the natural course of underlying independent organic heart disease". Also, we must not regard the symptoms of a cardiac neurosis as being caused by trauma to the heart.

The juvenile thorax is so easily compressed that damage may be done to the heart without a fracture of the bones. In more rigid chests, strong compression often fractures the ribs, which may in turn damage the heart and

pericardium. The electrocardiogram may show the changes usually associated with myocardial infarction or intraventricular block. Extrasystoles or even auricular or ventricular fibrillation may be seen, and so sudden death may occur. Usually these abnormalities disappear in a few weeks, but sometimes they persist, and classical angina may develop for the first time immediately after the trauma (Campbell).

Indirect Trauma.

Rupture of an aortic cusp may be due to indirect trauma. There may or may not have been previous aortic valve disease. The lesion causes the abrupt development of aortic incompetence. Likewise, rupture of an aneurysm, either of the aortic or of the ventricular wall, may be the result of a sudden great increase in pressure during some unusual strain.

Electric Shock.

Electric shock can produce many different disorders of heart rhythm, but especially extrasystoles, and also *S-T* segment and *T* wave changes in the electrocardiogram, and it may cause sudden death from ventricular fibrillation.

Occupational Infection.

An interesting case of a disease of the heart which can be regarded as due to employment was encountered by me recently.

A young man, a butcher by trade, who had old rheumatic carditis, was admitted to hospital with the clinical signs of early subacute bacterial endocarditis. The causal organism was found to be *Erysipelothrix rhusiopathiæ*, which is commonly encountered by butchers.

Arsenical Poisoning.

A less common form of heart disease in industry is that caused by chronic arsenical poisoning. The patient may die of heart failure. In such cases there have been found on post-mortem examination hæmorrhages in the epicardium and endocardium, especially in the endocardium of the left ventricle. In patients who live for some time there is found fatty degeneration of the liver, kidney and heart (Kerr).

Affection of the Coronary Arteries and Myocardium.

All the above-mentioned conditions are not likely to cause differences of opinion among medical witnesses, for the direct causal relationship between the injury and the heart lesion is well established. It is when we come to consider the question of whether or not strenuous effort, or long-continued laborious work, can affect the coronary arteries and the myocardium that we encounter much difference of opinion.

Friedberg states that "the evidence linking physical strain to coronary thrombosis is tenuous", and "in the majority of cases (95%) it appears without causal relationship to any physical strain".

A common story is that of a middle-aged man who, during or immediately after a strenuous effort, is stricken with a severe pain in the chest, and is found to have a myocardial infarction, confirmed by the electrocardiogram. It may be said that the infarction was bound to happen because his arteries were diseased, and that its relation to work was purely coincidental.

A. M. Master, while he points out that only a very few cases (2%) occur during strenuous effort, nevertheless considers that a prolonged effort may produce infarction without an occlusion in a coronary artery. The lesions in this latter instance are subendocardial, and not the typical lesion seen in coronary artery occlusion. It is difficult to believe that a worker could ever make such a prolonged strenuous effort, for he can voluntarily cease his effort if he is distressed by pain or difficult breathing. Since myocardial ischæmia causes pain, he should be warned in time.

Some observers have postulated that an atheromatous plaque is dislodged during the effort, and is carried onwards to block a smaller artery. Nobody, to my knowledge, has been able to demonstrate this, and this theory has now been abandoned.

Another theory is that during effort the blood pressure is raised enough to cause the *vasa vasorum* of the coronary arteries to rupture and produce a subintimal hæmorrhage, which bulges inwards to occlude the artery. The work of King shows that this does not happen. Also it has been stated that the intima of diseased coronary arteries on the surface of the heart before they enter the heart muscle may rupture during a strenuous effort, the result being either death or a locus for the development of a later thrombosis, and so may give rise to either death or coronary occlusion.

This conception merits consideration; but so far its truth has not been established. On the other hand, Winternitz injected diseased coronary arteries after death and found that even with high pressure (500 to 1000 millimetres of mercury) they did not rupture.

But what of the person who dies during or within a few minutes after a very strenuous effort? This is a difficult problem, for it is impossible to know what happens at the moment of death. An attractive theory is that the effort produces a severe ischæmia in a myocardium already poorly supplied with blood owing to coronary artery disease, and that this ischæmia is sufficient to produce ventricular fibrillation and death. This is quite likely to be true, although there is no certainty about it, for anginal pain should occur first. In the present state of our knowledge I feel that we must accept this view in compensation cases.

French and Dock, in a review of fatal coronary arteriosclerosis in young soldiers, related the histories of a number of young men who died suddenly after strenuous exercise. All had preexisting disease of the coronary arteries without symptoms. They came to the conclusion that "vigorous effort and the activities of early morning chores brought on the fatal attacks in over 50% of the cases". In some cases no thrombus was found, in others there were narrowing and atheromatous deposits. Infarction was not always seen. Evidently they consider that sudden myocardial anoxia was the cause of death. These are isolated examples and may have been coincidental.

Master and Jaffe call such a case one of coronary insufficiency and admit its causal relation to effort.

A workman who has a myocardial infarction while doing his ordinary work, which is not unduly laborious, may claim that his work is a factor in the causation of the infarction. In this case I consider that his illness is coincidental with his work and not caused by it.

Another type of claim arises when a man who undertakes heavy exertion, without any symptoms at the time, suffers from a myocardial infarction hours later—probably at rest at home. It is said that the building up of a subintimal hæmorrhage causes a blocking of the coronary artery some hours after the effort. Subintimal hæmorrhage is a part of the pathological process in atheromatous disease; but there is no support for this view that it is influenced by effort. The question becomes more difficult when we realize that the process of blocking a coronary artery (sometimes by thrombosis and sometimes without it) may have been going on for some hours or some days before the effort. Then the effort could be regarded as a coincidence. Nevertheless, it is possible to argue both ways, for we must admit that, although the causal connexion cannot be seen, it does not necessarily follow that there is no such connexion. I cannot admit a direct causal relationship until there is better evidence than we have at present.

Boas holds the opinion that "the syndrome of coronary occlusion may be induced by a non-penetrating injury to the chest or by unusual effort"; also that "when an unusual strain during work is followed by cardiac disability in the sense outlined and when this occurs in a person who has been previously well and free from symptoms while at work, it is proper to conclude that the

disability was induced by the work and is therefore compensable". This view is opposed by Master, who regards the coronary occlusion as coincidental. Phipps found that in 40% of his cases coronary thrombosis occurred under conditions of physical or psychical stress, or accompanying indigestion or exposure to cold, and that in 13% it occurred during exercise.

Sigler tells the story of a man, aged fifty-two years, who, while walking in a dark corridor, tripped and fell to the floor. He was unconscious for some time. In hospital he was found to have a fractured right fibula. An electrocardiogram taken later revealed the presence of a myocardial infarction. Since then he has been subject to recurring attacks of retrosternal pain on the slightest exertion. The author comments: "The case then, is one of infarction of the posterior wall of the left ventricle caused by injury to the heart. The infarction was undoubtedly due to coronary artery thrombosis which was precipitated by the injury." Evidently, the author has no doubt; but it seems to me that it is more likely that the man suffered from a spontaneous infarction, causing him to fall.

Recently I was asked to give an opinion about a man who had been given compensation in 1943 for damage to his heart while lifting a bag of coke. He died in May, 1951. Was his death the result of "injury" in 1943, or was it due to the natural progress of the arteriosclerosis? I expressed the opinion that his death was due to natural causes and not related to his work.

Frequently a claim for compensation is based on a history such as the following. A man, aged sixty-nine years, walked up a hill to catch a tram on his way to work. He was seen boarding the tram, appeared quite well, and spoke cheerfully to a friend. Four or five minutes elapsed before the tram began its journey. It made three stops. After the third stopping place the man died on the tram. I gave the opinion that death was due to heart disease and was not influenced by effort, for I could not understand how he could be quite well after walking up a hill if the effort had been harmful; nor could I think of any mechanism by which the effort could have caused death.

Primary Cardiac Overstrain.

Barber discusses the problem of primary cardiac overstrain, and considers that "this is a very exceptional event which may occur in an untrained person undertaking such exertion as rowing, or climbing or heavy lifting with the glottis closed. Structural damage has never been observed; only some type of arrhythmia with lessened functional capacity." He also observes that clinical signs are of little assistance in assessing the diagnosis of trauma.

I have observed arrhythmia after effort, which quickly came under control, and I did not regard the effort as damaging to the heart. I agree with him when he says: "There is no proof that a normal heart can suffer as a result of strain and to base a diagnosis on the probabilities of the history and the symptoms is unsatisfactory." Finally, he says that "it may be wise to admit the possibility of its occurrence". I have difficulty in admitting this.

Thus, because we do not know the whole truth, we are forced to admit this possibility and so the matter remains undecided. Most of us take the view that the healthy heart is not damaged by effort, and that acute dilatation of the healthy heart does not occur. S. A. Smith ably wrote about this matter in 1931.

If coronary artery disease is present, then effort may cause ischæmia of the myocardium and the patient will be stopped by pain in the chest and dyspnœa. In some rare instances the person may not be able to stop, and the myocardial ischæmia may, I assume, cause death. But in industry the workman can stop when he wishes to do so.

Pulmonary Fibrosis.

Another type of claim arises when a worker, who develops pneumonokoniosis and receives compensation for it, is found later to have ischæmic heart disease. He will complain of increasing dyspnœa and will be found to have enlargement of the whole heart and ultimately develops symptoms and signs of left and right ventricular failure; or, he may die suddenly from coronary occlusion. Can his new disability be related to pneumonokoniosis? Left ventricular failure is not caused by lung disease unless the latter is so severe as to lead to defective oxygenation of the blood, which hampers the myocardium. Thus death from coronary occlusion cannot be caused by pulmonary fibrosis.

It is well known that failure of the right ventricle occurs when there is left ventricular strain or failure. But in cases of pulmonary fibrosis and emphysema without heart disease, pulmonary hypertension develops, which causes right ventricular hypertrophy, and later congestive heart failure, which will lead to death even when the left ventricle and valves are normal. This is often an industrial disease. In 1937 Nemet and Rosenblatt, in a study of chronic pulmonary tuberculosis, found that, of 71 patients whose organs were later examined *post mortem*, no less than 33 (46·5%) had right ventricular hypertrophy without associated coronary artery and valvular disease or hypertension, indicating that the chamber had been working against an impediment in the lesser circulation. Thus, if a worker has pulmonary fibrosis due to his occupation, and this is followed by pure right-sided heart failure, he should be compensated.

The difficult cases are those in which pulmonary fibrosis exists and has been accepted as an occupational disease, and in which symptoms and signs of right-sided and left-sided heart failure are found and the autopsy reveals extensive coronary artery disease. If there is much pulmonary fibrosis with hypertrophy of the right ventricle, then we can regard the heart disease as having been aggravated by the subject's occupation. But if the fibrosis is slight and the coronary arteries are greatly diseased, the death is due to ischæmic heart disease, and so is not caused or aggravated by his employment.

I recently examined a miner, aged fifty-eight years, who complained of cough with sputum and increasing shortness of breath. He was thought to have heart disease in addition to some pneumonokoniosis. Two radiographs of the chest at an interval of two years showed "slight non-disabling pneumoconiosis" with no alteration in the amount of fibrosis in the two years. Clinical examination of the cardiovascular system revealed no abnormality. Fluoroscopic examination revealed right ventricular enlargement, and the electrocardiogram was normal. No bronchitis was present. This man had well-developed emphysema, and so I expressed the opinion that his disability was not caused or aggravated by his occupation.

If pulmonary fibrosis is extensive enough to cause anoxia, the heart will be affected by this. But by the time this happens the man will be showing signs of congestive heart failure in addition.

Cardiac Neurosis.

Cardiac neurosis is a well-known condition, the causes of which are numerous. Nowadays many people are heart conscious, so that many and varied symptoms are related to the heart. Over-solicitude on the part of the parents may be the beginning of a neurosis. An undue importance may be attached to a leaking valve; or the physician attending a child with rheumatic fever may be too gloomy, and so frighten both the parents and the child. His play is then limited too much and his whole life is disrupted, with the result that later he cannot adjust himself to employment in industry.

A healthy worker may sustain a slight injury which causes pain in the chest, and he himself, or a friend, or a first-aid man, may state that he has strained his heart. This view may be supported by his doctor; or it may be suggested to him by some remark, or by the fact that the stethoscope is kept longer at one place than another. Often minor variations in the electrocardiogram are given too much importance. This is a common mistake, and is a powerful factor in producing a neurosis. The following is a typical history.

A healthy man, aged thirty-four years, who had never been ill before, was using a crowbar digging heavily. He felt a sharp pain "across the heart" (referring to the left side of the chest). The pain eased with rest, but he noticed a "thumping of the heart". He rested at home for two weeks, during which time "there were a few missed beats of the heart", and the pain persisted. After a holiday he resumed work feeling well—but such symptoms as ache in the chest, shortness of breath, gripping in the throat, giddiness and numbness of the fingers bothered him from time to time. A complete investigation revealed a normal cardio-vascular system. There was tenderness in the chest muscles on the left side, and pain could be reproduced by strong action of the pectoral muscle. The diagnosis was strained chest muscles and cardiac neurosis.

A cardiac neurosis once established is very difficult to remove, and unless such a patient can be reassured and made to realize that his heart is normal, he will remain unfit for work. Thus we must be careful not to tell a patient that he has strained his heart. In fact, we should rarely mention his heart except to reassure him. If the physician has any doubt he can refer the patient for special investigation.

Congenital Heart Disease.

Congenital heart disease is rarely a problem in industry, for few such patients are fit to be employed. People with such lesions as patent *foramen ovale*, uncomplicated interventricular septal defect, patent *ductus arteriosus* and sub-aortic stenosis may have few or no symptoms, and lead a normal life. Much depends on the size of the lesions in the first three. These patients are liable to complications such as rheumatic cardio-vascular disease, and especially subacute bacterial endocarditis. This latter complication was present in my butcher patient, who has an interventricular septal defect in addition to rheumatic carditis.

It is well known that these patients may die suddenly, even though they are free from symptoms, so that a claim for compensation will arise if death occurs at work. But we have no evidence that work either caused or hastened death, which must be attributed to the nature of the disease itself.

Time does not permit me to deal with such questions as the employment of cardiac patients in industry, or with the methods of rehabilitation. Big industrial firms will in the future, I feel sure, have a cardiac clinic to advise the management about all such patients, as is done in the Altro Workshops in New York City (Jezer, 1953).

Conclusion.

In conclusion, many ideas about the heart which have been held for hundreds of years have been proved to be wrong, and we owe a great deal to the accurate observations of a general practitioner, James Mackenzie, who showed that the heart should be judged on what it can do and not so much on what is heard by the stethoscope.

Almost invariably, when a layman hears of a person who has heart disease he remarks: "He has worked too hard." Many men begin to slow down after the age of forty years in order to prevent heart disease or hardening of the arteries. In my opinion there is no justification for these views. I was pleased to read what Dr. Paul D. White said at the recent annual meeting of the American Medical Association:

> The general warning to stop all exercise at the age of forty seems to me ridiculous.

(Dr. White, at the age of sixty-seven years, still hunts whales.)

References.

BARBER, H. (1938), "Trauma of the Heart", *Brit. M. J.*, 1: 483.

BARBER, H. (1940), "Contusion of the Myocardium", *Brit. M. J.*, 2: 520.

BARBER, H. (1944), "Effects of Trauma Direct and Indirect", *Quart. J. Med.*, 13: 137.

BECK, C. S. (1935), "Contusions of the Heart", *J.A.M.A.*, 104: 109.

BOAS, E. P. (1939), "Angina Pectoris and Cardiac Infarction from Trauma or Unusual Effort", *J.A.M.A.*, 112: 1887.

CAMPBELL, M. (1939), "Angina Pectoris following Crushing Accident", *Brit. Heart J.*, 1: 177.

FRENCH, A. J., and DOCK, W. (1944), "Fatal Coronary Arteriosclerosis in Young Soldiers", *J.A.M.A.*, 124: 1233.

FRIEDBERG, C. K. (1950), "Diseases of the Heart", Saunders, Philadelphia and London.

JEZER, A. (1953), "Work Capacity of the Cardiac", *M. Clin. North America*, 37: 1667.

KEER, D. J. A. (1946), "Forensic Medicine", Black, London.

KING, E. S. J. (1952), "The Hæmodynamics of Subintimal Hæmorrhage", *Australasian Ann. Med.*, 1: 18.

LAWES, F. A. E. (1952), "Subacute Bacterial Endocarditis caused by Erysipelothrix Rhusiopathiæ", M. J. AUSTRALIA, 1: 10.

MASTER, A. M. (1945), *J.A.M.A.*, 129: 90.

MASTER, A. M., DACK, S., and JAFFE, H. L. (1937), "Factors and Events associated with Onset of Coronary Artery Thrombosis", *J.A.M.A.*, 109: 546.

MASTER, A. M., DACK, S., and JAFFE, H. L. (1938), "Post-operative Coronary Artery Occlusion", *J.A.M.A.*, 110: 1415.

MASTER, A. M., and JAFFE, H. L. (1952), "Factors in the Onset of Coronary Occlusion and Coronary Insufficiency", *J.A.M.A.*, 148: 794.

NEMET, G., and ROSENBLATT, M. B. (1937), "Cardiac Failure Secondary to Chronic Pulmonary Tuberculosis: Necroptic and Clinical Study", *Am. Rev. Tuberc.*, 35: 713.

PHIPPS, C. (1936), "Contributory Causes of Coronary Thrombosis", *J.A.M.A.*, 106: 761.

SIGLER, L. H. (1942), "Trauma of the Heart due to Non-penetrating Chest Injuries", *J.A.M.A.*, 119: 855.

SMITH, S. A. (1931), "Trauma of the Heart", M. J. AUSTRALIA, 2: 575.

WARBURG, E. (1940), "Myocardial and Pericardial Lesions due to Non-penetrating Injury", *Brit. Heart J.*, 2: 271.

WHITE, P. D. (1953), *Time*, June 15, page 42.

WINTERNITZ, M. C. T., and LECOMPTE, P. M. (1938), "The Biology of Arteriosclerosis", Springfield, Thomas. Quoted by Master, A. M. (1945), *loco citato*.

HEART DISEASE IN INDUSTRY.[1]

By ULRIC L. BROWN,
Sydney.

I AM to speak to you on coronary heart disease in industry. I propose to do so in the more or less simple terms that I would use in dealing with an actual case, and I shall avoid, as far as possible, the purely academic.

As most of you know, I am with the New South Wales Department of Railways and we have some 55,000 employees. I suppose I see each week, in new cases and follow-up examinations, an average of 20 patients with recognized coronary disease, and have to make decisions regarding their fitness for work and the type of work which each will be asked to perform.

As this disease bulks so largely as a cause of lost time, and as we have but limited time to give to individual patients, it is desirable that we have a good clinical knowledge of the condition and its pathology in order to recognize it in its classical and atypical forms, and to have concise, clear and practical rules for its management. As all this can be gained only from a full picture of the underlying pathology of the condition, I should like to indicate to you briefly my conception of this disease process.

The heart is a muscular organ obtaining its blood supply through the right and left coronary arteries, which come off the very beginning of the aorta and communicate directly with each other through major branches, thus providing the opportunity for collateral supply in the event of blockage in either artery. The coronary arteries are unique in that they tend to empty during cardiac systole and fill during cardiac diastole—a point worth remembering in assessing effort with its possible elevation of blood pressure as a factor in the causation of coronary occlusion. Each coronary artery branches and rebranches just like a tree, the terminal twigs of which represent the capillaries. Some of these, one type of *vasa vasorum*, end in the substance of the wall of coronary branches other than the ones from which they arose, and are thus far removed from their source and have a correspondingly low blood pressure.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 27, 1953.

The other type of *vasa vasorum*, the luminal capillaries, come off the lumen of a coronary artery and complete a short course in the substance of the wall of the same artery. These luminal capillaries are not unimportant in the medico-legal argument surrounding the significance of so-called subintimal hæmorrhages in the causation of coronary occlusion.

The pathological basis of coronary artery disease is atheroma and the degenerative changes and tissue reactions that ensue both in the wall of the artery itself, and in the myocardium supplied by the affected artery. The changes in the wall of the artery result in thickening, deformity and hardening of the wall at the expense of the lumen. There is progressive narrowing of the opening in the artery up to complete loss and obliteration.

The changes in the myocardium are a replacement fibrosis—the muscle fibres die and are replaced by scar tissue; but this occurs only with failing blood supply to the myocardium. It does not occur if an alternative blood supply is and continues available.

Such is only part of the picture of the pathological basis of coronary artery disease. If it was the whole, relatively few of us would live to enjoy a ripe old age. Fortunately for us, at the same time as our old coronary arteries that are affected by this disease of coronary sclerosis are progressively blocking up, new vessels are developing in the form of capillaries, and other less affected old communicating branches are dilating, the whole providing an alternative collateral circulation—and this opening up of new channels is likewise progressive.

Every day of our lives our old coronaries are a little more obstructed, but our collateral circulations are a little wider and admit a little more blood. We shall experience symptoms only if, at any time, the heart muscle lacks an adequate blood supply for the work it has to do. If this lack is relative—that is, if there is too little normal blood, or if there is a normal blood volume but the blood carries too little oxygen or too little hæmoglobin—there is relative myocardial ischæmia and we experience the pain of angina or coronary insufficiency. While this usually occurs on effort, it does occur at rest; but the pain of angina, in passing, leaves the myocardium exactly as it was before unless the duration of the ischæmia and anoxia is so prolonged that irreversible change ensues and we have then the scattered, pin-point infarction of coronary insufficiency.

Let me say a word or two about *angina pectoris*. It is well to remember that when we decide that an employee is suffering from this condition, we also say that he is the victim of coronary sclerosis and a candidate for any of the catastrophes that characterize this disease, and for the remainder of his industrial life his employment will be directed, and in many cases restricted, possibly with loss of trade classification and income.

In the classical description, the pain of angina is substernal; but it may occur anywhere about the chest, arms or neck, it comes with increased heart action, it tends to compel rest, and, with the quieter heart action which ensues, it gradually passes off. With such a classical picture one hardly neds an electrocardiogram for possible confirmation—and I say possible, for many of such tracings show no abnormality to give evidence of the sclerosis.

When *angina pectoris* is suspected, direct your inquiries to the man's ordinary daily activities. You will find that on rising he has a restricted capacity for effort; he may feel the pain or discomfort while preparing for work, or on walking to his means of transport, especially if there is a set of railway steps on his path or if he has need to hurry. Often he will tell you that as the day advances he feels much freer about the chest, has a better capacity for effort and manages his walk home quite comfortably.

I suggest that a possible explanation of this very common observation lies in the opening up and filling of the collateral blood vessels by the improved blood flow that comes with effort. I do not mean by this that a quite new collateral network suddenly comes into existence (for more or less the same history repeats itself each morning), but rather that the vessels feeding a major collateral vascular network are themselves adequately fed only when the coronary blood flow gains a certain head of pressure and velocity, and thereby opens up and fills this existing collateral network and thereafter maintains a full circulation through it.

It is in the atypical case of suspected angina that we need to be most searching in our inquiries, and mistakes will be made both ways. I recall cases proven to be angina such as the following:

For several months the pain occurred only in a limited area about the inner aspect of the left elbow. It was an effort pain produced and reproduced by the same relative effort, even though the arms did not share in the exertion of the exercise. Some time later this man suffered an occlusion and the pain of the onset was limited to his left elbow; but, unlike his previous experience, it did not abate when he rested. Some twenty-four hours later he experienced sudden severe substernal and lasting pain, with the shock and collapse of a major coronary episode.

Another man complained of pain between the shoulder blades. He had been certified as having spondylitis of the cervical and thoracic parts of his spine. His, too, was an effort pain, more readily produced in the morning than later in the day. Inquiry indicated that it had been habitual for him for some months to mount steps more slowly, for to do so at his former pace invariably produced the pain. I thought in his case that if the pain was that of spondylitis, cervical and thoracic, it would have a relationship to posture, and to trunk and head movement rather than to walking. In his electrocardiogram the *T* wave in Lead I was flat and there was a negative *T* wave in CF_4; he was not hypertensive. Within some twelve months he suffered a major posterior occlusion.

Perhaps the pain of spondylitis that might be confused with angina would be more diffuse, would tend to radiate into neural zones inappropriate for cardiac lesions, would be postural and would be related to a particular movement rather than to general exertion.

Pectoral muscle strain, intercostal strain or fibrositis, painful left shoulder conditions and contused rib cartilage will all present difficulties at times in distinguishing them from *angina pectoris*.

Again careful inquiry is helpful, particularly with regard to the initial onset. Consider whether or not the injury, if one is alleged, was competent to cause any of the foregoing. An act of straining, pulling or lifting commonly causes muscle strain and so-called "contused rib cartilage", but often, too, gives a workman his first attack of angina.

As an aid to distinguishing between them, devise an exercise or movement that puts the part complained of on the stretch and produces the pain. After a rest and when the pain has passed off, put the patient through a further exercise which, while putting an equivalent demand on the heart, imposes no strain on the site of the pain. If the pain occurs, it is probably anginal; if not, it is probably one of the former, and, of course, of much less moment in regard to future employment.

Localized tenderness, and pain on breathing or deep breathing, stamp the pain as arising in the chest wall and completely dissociate it from the heart.

Angina pectoris, then, has for its basis a relative cardiac ischæmia—an inadequate blood supply to the myocardium relative to the demand made upon it. If now the blood supply to an area of myocardium fails altogether and no alternative blood supply is available, that area of myocardium must die, and we have myocardial infarction; either the blocked vessel is occluded by the atheroma *per se*, or the blocking is completed by a thrombus. The clinical episode marking such an event is certified as a coronary occlusion or thrombosis or myocardial infarction.

With the management of this phase the industrial medical officer will have little to do. His responsibility is mainly to recognize the event and institute effective management should the attack occur at work.

Once the condition has been diagnosed or merely suspected, place the victim at rest, assure warmth and

comfort and transfer him by ambulance to the nearest hospital. I always send an accompanying letter by the ambulance officer to be handed to the casualty surgeon, in which I briefly set out any facts regarding his medical history and his attack that are known to me. Remember an occlusion may be painless. You may be called to see a workman who has collapsed; he is in a state of more or less severe shock; he may or may not have pain; he may or may not have felt faint or giddy.

Examination of such a man, which of necessity may be brief and cursory, may show that he looks a "coronary" type; his pulse may be soft, according to the degree of shock, and fibrillation may be present. I have noted how frequently this arrhythmia is associated with occlusion, and how occasionally it is the only sign or basis for symptoms —so much so that I deem it wise to assume that fibrillation coming under notice for the first time has for its basis coronary artery disease with acute recent change until, from the history or subsequent consideration, I can discard this possibly more serious view.

Mistakes in attributing collapse to coronary artery disease will certainly occur, and I mention four such in my own experience—namely, cases of hæmorrhage in a peptic ulcer, of spontaneous pneumothorax, of a hæmothorax in association with a fractured rib, and of acute hæmorrhagic pancreatitis. It will be appreciated that a prior knowledge of the employee's medical history must always be helpful, and such medical records can always be prepared and kept available by every industrial medical officer, as his potential patients are limited to the employees of the plant which he serves.

The next step, then, the man having been treated and reporting back for work, is to determine his fitness for work. In the first place allow ample time for recovery from the effects of an acute recent occlusion; I tend to think in terms of not less than three months. The dangerous period following the occlusion is the first three weeks after the occlusion, because of softening of an infarcted area giving rise to thinning or even rupture of the heart wall and embolism from separated mural clot.

I consider it bad treatment, when an acute coronary occlusion is suspected, to tell a patient to have a few days' rest and then report to a hospital with a letter you have given him to have an electrocardiographic examination. Apart from the catastrophic possibilities related to such activity, I am quite sure that inadequate rest in the presence of acute recent coronary blockage is a fruitful cause of subsequent persistent and disabling angina.

Ample time must be allowed for the development of the collateral circulation, and this is estimated by the man's capacity for comfortable effort. No matter how free from effort, mental and physical, a man's work may be, he should not be permitted to resume until he can prepare himself for work and go to and from his job in comfort, or with at most minimal discomfort. He should be able to walk comfortably on the level, and to mount a set of railway steps at a slower than walking pace without more than a little shortness of breath or slight anginal discomfort. He should not be permitted to resume if he has angina or shortness of breath on less effort, unless some other factor such as economic family stress compels such action. In assessing capacity for effort, I make no use of planned exercise tolerance tests, all of which contain an element of nervous upset for the man which would not operate to complicate the purely physical demands on his heart made by his work.

In my pre-placement examination, there is one sign I am very careful to look for—namely, *pulsus alternans*. This is the surest sign I know that all is not as well with the man as it seems. I may have an opportunity later of enlarging on this view.

Having decided then that he may return to work, I give him a clear picture of the pathology of his condition along the lines indicated in the early part of this talk. I stress the opening up of new blood vessels, and how much better off we are than is the case with corroding water-pipes; how the patient is possibly better off having had his occlusion than is the man who, though a candidate for one, does not yet know it and imagines himself to be a very fine fellow; how the man who has had an occlusion and recovered from its immediate effects must already have his collateral circulation well developed or he would have died. I point out to him that as the blocking up of his old arteries and the opening up of the new ones are both progressive processes, their relationship to each other must vary from time to time, and that he will have symptoms whenever his heart muscle lacks an adequate blood supply for what it has to do; that he must expect to have days when he will not feel so well as on others; that he is not to be unduly worried on such occasions, but must steadily apply the rule that he will do nothing that he cannot do comfortably; he must be his own doctor and remain the sole judge at the time of whether or not he shall get out of bed, or go to work, or if he is at work, whether he will continue if he does not feel equal to his job. I may point out that you, as doctors, in supporting such men in their decisions against a possibly unsympathetic authority, need have no fears of malingering or abuse of privilege on their part. Remember that a man who has had an occlusion in these informed days has been down into the Valley of the Shadow, a salutary experience. Furthermore, these men are of such an age group that they have acquired skill in their work and a pride in their output. If you have informed them thoroughly on the work aspects of their disease, neither their skill nor their output will suffer. It does not surprise me that carefully prepared statistics show that the work output of such men is 2·5% greater per man-hour than that of healthy controls, which more than compensates for their higher lost time rate of two and a half days per year.

So they are warned to do nothing that they cannot do comfortably, but to remain active within their limits of comfortable activity.

I stress that they can do in safety anything that they can do in comfort. Finally, I explain to them that a further attack may occur at any time and may be more severe than the one they have had—that sudden death may occur at any time, day or night, at work or at home, during activity or at rest; but I lessen the frightening effect of this realization by pointing out how much more likely this is to occur in a person who has not developed his collateral circulation, and who is unaware that he has the disease and continues his normal way of life, probably eating and drinking and living to excess. I convince him that the disease is as common as grey hair, and that of all the people who have this disease it is only a very small percentage indeed that are victims of sudden unexpected death, and that he, in his understanding of the condition, is in an excellent position to minimize the risk of a calamitous happening. He is warned, however, that a disabling turn may occur at any time, and because of this he must never be in a position such that should a turn occur in which he cannot help himself, there would be further risk to himself or to others. He is advised that should there be a return of his pain, or should his pain come with less effort, or should he contract influenza or any other infection, he is to cease duty at once and report for examination or go home to bed and send for his doctor.

After his return to work I reexamine him in one month and thereafter every three months until his condition appears stabilized, when the intervals are increased.

The type of work he is allowed to perform is determined by this pre-placement examination and by a knowledge of his working conditions.

He is not allowed to take up duty until he can get to and from his job comfortably, and he is allowed to do anything that he can do without discomfort, provided that the occurrence of an incapacitating turn would not endanger himself or others.

If possible, I prefer him to resume on his normal duties—for this avoids economic loss and changed working conditions; he remains among his former workmates, and under known authority, and, if his name among his work-

mates is good, they may be depended upon to ease the going for him. This also bolsters his confidence in himself and carries reassurance. If you are in any doubt about the full extent of his work, ask for a statement of his duties, and if possible see the work for yourself. In a small plant it should be possible to discuss the matter with his foreman and make him well informed and interested, and to enlist his sympathies by reminding him that he may even be the next victim.

In large industrial undertakings, where many men are employed and you possibly work through a rehabilitation officer and cannot discuss the man's duties with his foreman, it is necessary to certify that he is fit for non-strenuous duties that he can do without discomfort and such that the occurrence of an incapacitating turn will not endanger himself or others.

Unfortunately, in many unions, any medical restriction in a man's working will cause him to lose certain privileges, among them being the right to work overtime, and he can even be forced out of his usual place of employment if a fellow unionist protests against his inability to do all the duties of his trade classification. You may sometimes overcome this difficulty by allowing him to resume his normal work, but arrange that he does the less heavy parts of it by assuring that he has a better than average and dependable assistant or labourer or mate (as he is generally termed). Inspection of his job may show that there are but one or two strenuous efforts in his whole working day, and the installation of a crane, a hoist or a conveyer will obviate these altogether, or all that is necessary is merely the realignment of plant to reduce walking and carrying. Remember the men who call for such extra thought and inquiry are skilled men whose loss to industry can be ill afforded and whose pay margins are appreciable. I should consider it bad management and social blundering to provide such men with an unskilled light duty job carrying the basic wage.

Remember, too, that his condition may grow worse at any time and retirement prove the only course, and his retirement leave and privileges may be appreciably affected if he continues in the lower grade indefinitely.

For these and other reasons, you may prefer to have the employee retire rather than return to work. I am sure you would have a profound reluctance in asking any salaried man or highly skilled tradesman to revert to the position of lavatory attendant or yardman in his former plant. If one of our functions is to allay a man's fears and resolve his anxieties, it is far better that we should support his retirement and advise him to seek suitable selected work outside, part-time if necessary, or develop a paying hobby to supplement his retiring allowance.

Remember, too, in this connexion, the Service pension or "burnt-out-digger's pension", available through the Department of Repatriation to Service personnel of World Wars I and II.

In the foregoing I have not dealt at length with placement or rehabilitation, but have merely indicated broad, general principles which I use for my own guidance. In the Department of Railways we have some 55,000 employees, many living under camp conditions, often remote from medical aid on some far-flung rail link; the majority are concerned with the safe working of trains and with track maintenance. The incidence of this disease among them, I feel, is the same as that for the general population, and their care, placement and subsequent follow-up, while often presenting individual problems, offer no difficulty that cannot be solved by adherence to principles and by knowledge of working conditions.

Our workshop employees, refreshment room staff and some clerical staff are comparable, as to working conditions, with those in industry generally. So far as these are concerned, in placement I have in mind the capacity of the individual for comfortable effort against the physical demands of the work to be performed—and the risk to him and to others arising out of his work should an incapacitating turn occur—and the legal responsibility of the employer.

I do not allow him to work near open machines, or in close proximity to traffic or moving vehicles; he is not allowed to work at open vats, or to drive cranes, or work off ground level. The last-mentioned restriction may be relaxed a little to permit special placement. So far as weights to be lifted are concerned, I am particularly careful to know how many times he is required to lift them in a shift, and whether the lift is a clean one from ground or waist level, or whether the article has to be pulled from a heap rather than be taken from a stack. For weights to be lifted from time to time in a shift I tend to set a maximum of 20 to 30 pounds' clean lift. When the lifting is being carried out by gangs of men and their output is conditioned by a steady, even, though relatively small contribution from each, I prefer to say that a coronary subject is unfit for such work, as his disease is such that he will have his good days and bad days, and he cannot be expected to give a steady, dependable contribution of effort in gang labour.

I do not place such a man as a watchman, though this seems to be a general rule in industry, for a watchman has a territory to cover, stairs to climb (and these may be steep and the climbing arduous), a bundy to punch at prescribed times (and often these clocks are located in places difficult of access in order to assure that such remote areas will be regularly inspected); consider, too, the possible physical and mental stress consequent on surprising an intruder.

I do not allow a coronary subject to drive a vehicle on the job, because I do not know when he may have his next attack, or how severe it may be. If he owns a motor-car, I advise him not to drive it. I remind him that it is hardly fair, to say the least of it, for the employer to go to the trouble of finding him non-strenuous duties throughout the working week, if in the week-end he applies himself to the strenuous work of jacking up a motor-car to change a tyre in a difficult location. I impress on him that the risk of a sudden incapacitating turn, in which he may find himself quite helpless, even in stopping the car, is an ever-present threat no matter how well he may feel; and if a coronary subject is at the wheel of a motor-car he is a dangerous menace to himself, to the occupants of the motor-car and to other road users.

The problem of whether or not we shall allow a coronary subject to drive a motor-car confronts us in industry, as the *Workers' Compensation Act* covers a worker on his periodic journey to and from his work subject to the fulfilment of certain conditions, and many workers use their own motor-cars. While it is most desirable that we do our utmost to reduce such an employee's travelling time and assure, as far as possible, his comfort on his periodic journey, I do not feel that I should allow him to assume these extra risks, nor allow his employer to face the expense of defending a subsequent court action, the successful defence of which could well prove most difficult when one considers all the external circumstances that could be invoked to support a claim that the man's attack had more than a temporal relationship to the act of driving a motor-car.

Rather than unnecessarily add to the exacting labours of our already overworked Commission judges, and having in mind my protective functions as an industrial medical officer, I prefer to obviate all such risks by not allowing the coronary subject to drive his motor-car on his periodic journey to and from his work.

Conclusion.

In these remarks on coronary disease in industry I have endeavoured to express some views on management arising out of my own experience. It has proved to be a wandering statement, and, in conclusion, I would like to emphasize these points:

Coronary disease is a common disease and yearly robs industry of possibly one-quarter of all those who suffer an acute manifestation of the disease in the form of persistent angina, acute recent occlusion, abnormal rhythm or heart failure.

Its greatest incidence is in the early fifties, when men equipped by experience and training should yield their greatest contribution to industrial progress, and any avoidable loss must be eliminated.

Every case must be individually assessed, and you must steadily avoid the far too common and equally ignorant assertion that the manifestation of coronary disease marks the end of a man's useful working life. Remember, a man may do in safety anything he can do in comfort, and he is well advised to remain active within his limits of comfortable exertion, rather than sink into himself and lead a life of invalidism. If he remains active, I believe that the development of the collateral circulation is assisted by the increased blood flow which accompanies effort, whereas it is probably hindered by the stagnating blood flow of ill-advised rest.

Give the patient a clear picture of his condition, and let him be his own doctor.

As industrial medical officers, your remaining responsibility to the man is to adapt the job and its attending circumstances to the work capacity of the individual if he is to continue in employment, and be just as informed and helpful in your advice covering all aspects of retirement if his working days are over.

PERFORATED PEPTIC ULCER AT THE ROYAL NORTH SHORE HOSPITAL OF SYDNEY FROM SEPTEMBER, 1949, TO JUNE, 1953.

By THOMAS F. ROSE,

Consulting Surgeon, Hornsby and District Hospital; Honorary Surgeon, the Royal North Shore Hospital of Sydney; Relieving Surgeon, Department of Repatriation, Sydney; Tutor in Surgery, University of Sydney.

(From the Department of Surgery, the Royal North Shore Hospital of Sydney.)

FROM September, 1949, to June, 1953, inclusive, 62 patients with a perforated peptic ulcer were admitted to the Royal North Shore Hospital of Sydney—an admission rate four times higher than that prevailing in the previous decade, during which only 44 such patients were admitted (Rose, 1950).

The ulcer was duodenal in 41 of the patients and gastric in 20, whilst in one the position was never determined.

An attempt was made to classify each ulcer as acute or chronic, depending on the length of history prior to perforation and the appearance of the ulcer at operation. When autopsy or later gastrectomy was performed, microscopic findings were used as well. A history of less than one month's duration was perhaps somewhat arbitrarily decided upon as characterizing an acute ulcer. Some patients had no previous symptoms at all.[1]

The Methods of Treatment Used in this Series.

Thirty-six patients with a perforated duodenal ulcer and 18 with a perforated gastric ulcer were treated by the conservative operation of simple suture (with or without an omental graft), assisted by tube suction, intravenous therapy and the exhibition of antibiotics.

Of the remaining eight patients, five with a ruptured duodenal ulcer and one with a "peptic" ulcer were treated by aspiration of the stomach only (assisted, of course, by intravenous therapy and the antibiotics with adequate X-ray control).

Two patients, each with a ruptured gastric ulcer, had no treatment for the original perforation, only for its later complications.

Mortality.

There were five deaths in this series of 62 cases of perforated ulcer—a total mortality rate of 8%.

Three of these deaths occurred among the six patients treated by aspiration only, and one of the two untreated patients died.

One of the 54 operation patients died—a mortality rate of 1·8%, which is a considerable drop from the 14% operative mortality rate of the previous five years in this hospital.

Conservative Operation.

Perforated Duodenal Ulcer.

Of the 36 patients with perforated duodenal ulcers treated by conservative operation, 21 were male and five were female. The ages of the former varied from twenty-one to seventy-nine years, and of the latter from thirty-five to seventy years.

The ulcer was situated on the anterior aspect of the first part of the duodenum in each instance. It was chronic in 28 males and three females, whereas in only three males and two females was it acute.

Twenty-eight patients were operated on within eight hours of perforation (20 in from two to six hours). Owing to delay in admission to hospital, none of the other eight were able to be operated on before twenty-four hours after perforation. In four of these cases suture was not performed until forty-eight hours, sixty hours, seventy-two hours, and seven days respectively had elapsed after perforation.

In only one case was the perforation a second perforation, and as previous operative details were unavailable, it is not known whether the same or another ulcer was involved.

Only one patient had a simultaneous hæmatemesis from his chronic ulcer.

One male patient, aged seventy years, had had his perforation for a week, during which time he even swallowed solid food. In spite of general peritonitis and coincidental retention of urine due to a prostatic adenoma, he recovered uneventfully with suture of his perforation and a suprapubic cystotomy. He died eight months later from uræmia, having suffered no further ulcer symptoms.

Morbidity and Mortality.—Only five patients (four males and one female) had major complications; all had had chronic ulcer perforations, and all were in the age group of forty-five to seventy years. Two of these were operated on within six hours of perforation; one suffered right basal atelectasis, and the other wound dehiscence. One patient operated on twenty-four hours after perforation had right basal pneumonia and wound dehiscence. Two patients, one operated on sixty and the other seventy-two hours after perforation, each had a subphrenic abscess requiring drainage, the latter having general peritonitis in addition. The patient who died was a male, aged sixty-one years, with a chronic duodenal ulcer. This had been perforated for forty-eight hours and he had general peritonitis. He died on the tenth day with wound dehiscence and a terminal hæmatemesis. Autopsy confirmed the chronicity of the ulcer.

Perforated Gastric Ulcer.

There were 18 patients with a perforated gastric ulcer who were treated by conservative operation, nine being males aged from thirty-one to seventy years, and nine being females aged from twenty-one to eighty years.

Of the males, three had acute and six had chronic ulcers. Of the females, five had acute and four had chronic ulcers—that is, there were eight acute and ten chronic ulcers.

[1] It is now the custom to try to classify perforated peptic ulcers into acute and chronic ulcers (and even subacute) because the acute ulcer is said to have both a better immediate and late prognosis (Gilmour, 1953).

However, only very roughly does the length of history coincide with the operative and histological findings. One patient, for instance, may have no previous history and yet have an ulcer both macroscopically and microscopically chronic. Another patient with a long history of ulcer symptoms may have a succession of acute ulcers rather than one continuous chronic one. At operation, too, it is difficult sometimes to tell the state of the ulcer after it has perforated owing to the superadded surrounding inflammatory changes.

The position of the ulcer was prepyloric in 14 patients, on the anterior surface of the body of the stomach in two, and on the distal part of the lesser curvature in two. There was no correlation between the age or sex of the patient and the position of the ulcer or its type.

The diagnosis of perforation in these perforated gastric ulcer cases seemed to be more difficult than in the duodenal ulcer cases, because many of them were referred to hospital so much later. Consequently, whilst six were able to be operated on from one to six hours after perforation, three could not be operated on until between twelve and twenty-four hours after rupture of the ulcer, four between twenty-four and thirty-three hours after rupture, and five later than thirty-three hours after rupture. This tardiness in diagnosis may be due to the fact that many ruptured gastric ulcers start off with very equivocal symptoms as compared with the more or less clear-cut clinical picture of the ruptured duodenal ulcer.

One patient had a concomitant hæmatemesis, for which simple suture sufficed.

Two patients, each with a chronic duodenal ulcer known to have been present for many years, developed a perforation, one of an acute prepyloric ulcer and the other of an acute ulcer on the body of the stomach.

Mortality and Morbidity.—There were no deaths from the conservative operation for perforated gastric ulcer. The morbidity rate was low, only three patients being involved. All were in the older age groups and had long-standing perforations (twenty-four to forty-eight hours). One patient had a wound infection and acute bronchitis which caused a later wound herniation requiring nylon weave repair. Two patients had wound infections, and one of them also developed a pelvic abscess.

Aspiration Treatment.

All the six patients who were treated by aspiration had radiological evidence of gas under the diaphragm on their arrival, and in all cases except one (diagnosed as a peptic ulcer) the presence and situation of the ulcer were confirmed later by a barium meal examination or at autopsy.

These patients are divided into three groups, each containing two patients, according to the reason for their treatment by aspiration.

Patients Whose Symptoms Were Already Subsiding on Entry to Hospital.

A male patient, aged seventy-one years, had an acute duodenal ulcer which had been perforated for ninety-six hours. The symptoms subsided and the patient was discharged from hospital on the twenty-first day.

A female patient, aged fifty-three years, had a chronic duodenal ulcer which had been perforated for twelve hours. Her symptoms subsided, and she was discharged from hospital on the twenty-first day.

Patients Judged Too Ill or Too Frail for Operation.

A bedridden female patient, aged seventy-eight years, had a chronic duodenal ulcer which had been perforated for four hours. She was able to be discharged from hospital in twelve days, even though her convalescence was complicated by collapse of the base of the right lung. However, the day after she went home, she died from what seemed clinically to be a reperforation.

A male patient, aged sixty-five years, had a chronic "peptic" ulcer. (On his admission to hospital he had the characteristic subdiaphragmatic gas bubble, but for some reason a later barium meal examination was never carried out.) This ulcer had perforated forty-eight hours prior to his admission to hospital. In the previous nine days he had had repeated cardiac infarctions, substantiated by electrocardiographic examination. He was treated by aspiration, and all went well until the ninth day, when he had a severe melæna requiring blood transfusions. He then developed a subphrenic abscess, which was evacuated successfully in the fifth week. A follow-up fifteen months later showed that he was very well, with no further ulcer symptoms.

Patients who Refused Operation but Accepted Aspiration Treatment.

A male patient, aged thirty-three years, had a chronic duodenal ulcer, which had perforated forty-two hours before his admission to hospital. He died five weeks later as the result of sepsis from general peritonitis. Autopsy revealed that the perforation was unhealed and communicated with a large abscess in the midst of dense adhesions in the right subhepatic space. Histological examination confirmed the chronicity of the ulcer.

A female patient, aged thirty-two years, had a duodenal ulcer which had perforated eight hours before her admission to hospital. She died of general peritonitis eleven days later. Though she had had no ulcer symptoms prior to perforation, autopsy revealed a chronic ulcer, the presence of which was confirmed histologically. It was interesting to note that the ulcer was shut off from the general peritoneal cavity by recent inflammatory adhesions save for one small sinus, which was the cause of her peritonitis and death.

No Treatment.

There were two patients in the group given no treatment. The first was admitted to hospital one week after the ulcer had perforated, and the condition of the second was undiagnosed until peritonitis supervened. The details are as follows.

A female patient, aged fifty-eight years, was admitted to hospital on the seventh day of her illness, having suffered from perforation of an acute gastric ulcer situated on the anterior aspect of the body of her stomach. This formed a large localized perigastric abscess, which was drained, with a successful result.

A female patient, aged forty years, had a previous history of nine weeks' epigastric pain and repeated mild hæmatemeses. However, she was suffering from *Streptococcus viridans* septicæmia on her admission to hospital, with no symptoms or signs referable to the abdominal viscera until she developed general peritonitis and a subphrenic abscess from an unsuspected perforation of a chronic gastric ulcer on the anterior aspect of the stomach. Death ensued after six months' illness from sepsis, in spite of drainage of the abscess. Autopsy revealed that the perforated ulcer was still open into the general peritoneal cavity, being only partially closed by adhesions.

Discussion.

The Conservative Operation of Suture.

From this small series, then, the conclusion may be drawn that the conservative operation of suture is safe, being attended by very low mortality and morbidity rates, even when one is dealing with elderly patients with a late perforation. This operation has the advantage of simplicity and ease in performance, so that it does not require the resources of a specialist clinic.

The present low mortality and morbidity rates of this operation are not necessarily due to the fact that patients are in better condition and were received earlier after perforation, because some of them still come in an extraordinarily long time afterwards, especially those with a perforated gastric ulcer. They are due to better parenteral therapy and gastric suction both before and after operation, and to the antibiotics.

Aspiration Therapy.

The method of aspiration therapy alone is a blind method, and even if the diagnosis is correct, one can only guess what is happening inside the abdomen. There is only one indication for its usage, and that is for those patients who are obviously settling down on their admission to hospital. Even these must be carefully watched, for even after an apparently successful outcome one patient in this series suffered reperforation with a fatal result.

The method has only one advantage—namely, that the patient avoids an operation. Even so, chest complications, once thought to be the prerogative of the operation, may still occur.

Its disadvantages, apart from the fact that the stay in bed is longer and frequent X-ray examinations are necessary, are as follows: (i) the diagnosis may be incorrect; (ii) there is a grave risk that the perforation may not heal—nature is not always so successful as the surgeon in closing these ulcers; (iii) the mortality rate is high—8% to 10% in reported series (Gilmour, 1953).

Partial Gastrectomy as an Emergency Treatment for Perforated Peptic Ulcer.

There has arisen again a tendency to perform partial gastrectomy for ruptured peptic ulcer, in order not only to relieve the perforation, but also to cure the ulcer at one and the same operation.

Originally, some ten to twenty years ago, Continental surgeons performed this operation on all patients with a perforated peptic ulcer as a routine procedure, because they stressed the high mortality at that time of the conservative operation as an argument for excision of the stomach which, in their hands at least, appeared to have no mortality, little morbidity and few after-effects. Now, with the low mortality rate of the conservative operation (1% or less), this argument is vitiated because that of even an elective gastrectomy is higher (nearer 4%).

Surgeons now who use this operation do so only on those peptic ulcers which they judge to be chronic. Nuboer (1951) states that all ulcers which perforate are chronic, and consequently he performs partial gastrectomy on all perforated ulcers as a routine procedure. However, other authors do not agree that all perforated ulcers are chronic. In Gilmour's (1953) series of 206 perforated peptic ulcers, only 87 or 43% were chronic. In the series of 62 cases recorded here, however, 46 were chronic. It would appear, therefore, that the ratio of acute to chronic ulcers differs according to the country and to the individual surgeon.

In order to justify the use of the more formidable operation of partial gastrectomy rather than that of conservative suture, its protagonists must show three things—namely, (i) that its mortality is lower than or at least equal to that of the conservative operation, (ii) that all chronic peptic ulcers treated by conservative suture when perforated cause later symptoms or complications so severe that an emergency gastrectomy is warranted to prevent them, and (iii) that the results of emergency gastrectomy are always as satisfactory and free from after-effects as Nuboer (1951) and many other Continental surgeons imply.

None of these suppositions is correct. To deal with them *seriatim*, in the first instance it has already been shown that the mortality rate in recent series of perforated peptic ulcers treated by the conservative operation is lower than that of partial gastrectomy.

Secondly, though a perforation is but an incident in the life history of a peptic ulcer, many of these even chronic ulcers, especially duodenal ulcers, produce few symptoms prior to perforating and few after the perforation has been sutured, even if the after-care of the patient when discharged from hospital has been haphazard. Naturally, if the after-care is carefully supervised, then the outlook is better. Though Gilmour (1953) points out that the prognosis of the acute ulcer which has been sutured is much more satisfactory than that of the chronic ulcers, all of which will sooner or later relapse as time goes by and require further medical and possibly surgical treatment (Illingworth, Scott and Jamieson, 1946), nevertheless, as may be shown by the admittedly brief follow-up of the ulcer patients in this review, even the patients with chronic ulcers may enjoy some years' freedom from symptoms after perforation.

Seventeen of the patients with sutured perforated duodenal ulcers were followed up for periods varying from two to four years. Nine (five with chronic ulcers at the time of perforation) had no symptoms at all during this time. Five (four with chronic ulcers) had symptoms of mild "indigestion" only, two having had some spasmodic treatment in the form of diet and alkaline mixtures. In one of these last-mentioned cases an opaque meal examination three years after perforation revealed that the ulcer was still present and would require more intensive therapy. One patient, whose ulcer was acute at the time of perforation, had a hæmatemesis one year later. After medical treatment he was well one year later. Two patients, both with chronic ulcers, had a partial gastrectomy, one five months later for a severe hæmatemesis, and the other only a month later for pyloric stenosis.

Seven of the 18 patients with perforated gastric ulcers were followed for the same length of time. Six patients (two with chronic ulcers) had no further symptoms. One patient with an acute ulcer at the time of perforation was well for two years and then had a mild hæmatemesis; after medical treatment for this she has remained well for the six months to date.

The duration of the follow-up examination in these few cases is admittedly short, and the picture will undoubtedly become worse as time goes by. Nevertheless, many of these patients have been living normal lives for at least two to four years after the operation of simple suture for perforated peptic ulcer, even though they still possess a stomach.

Thirdly, contrary to the views expressed by the emergency gastrectomists, who lead one to believe that once a gastrectomy is performed all the patient's troubles are over as far as his stomach is concerned, we know that even an elective gastrectomy is followed by early post-operative morbidity and later disconcerting sequelæ. Early in the piece, especially after gastrectomies for perforations, a duodenal fistula is not uncommon, and even if it is only a terminal and not a lateral fistula, it is nevertheless a most uncomfortable burden for the patient. Again, apart from the different varieties of the so-called "dumping syndrome", there are the post-gastrectomy food deficiencies (post-gastrectomy marasmus), deficient fat absorption and failure to gain weight, and the attacks of extreme lassitude which beset some patients (Editorial, 1950). Again, Moroney (1953) states that after the usual partial gastrectomy as practised today, there is at least a 4% ulcer recurrence rate, and because of this and the other woes which may beset the post-gastrectomy patient he has devised replacement of the lost stomach tissue by the transverse colon, with the usual spectacular results of a new operation.

Lowdon (1952) makes a very pertinent observation when he points out that a patient with only vague dyspeptic symptoms before a perforation may not be pleased with the new trail of symptoms which may follow a gastrectomy. A patient suffering from the pain of a ruptured ulcer is hardly in a position to discuss the pros and cons of a gastrectomy with his surgeon, and he may later regret the surgeon's choice.

If these facts are borne in mind, then, the correct treatment of a patient with a perforated peptic ulcer is to perform suture of the ulcer. The patient must then be carefully followed up and treatment given as required when his condition has been assessed. He may require further medical treatment or even an elective gastrectomy, which can be performed on a patient both physically and mentally prepared for it.

Are there any indications for the performance of emergency gastrectomy in the presence of a perforated peptic ulcer? Certainly, though they rarely arise. They are as follows: (i) the suspicion that the ulcer is carcinomatous; (ii) simultaneous hæmorrhage from the ulcer (two patients in this series whose ulcers bled and perforated at the same time did very well with simple suture); (iii) a large chronic ulcer whose edges are difficult to close (though even here an omental graft will close the perforation successfully).

The fact that one has a specialist anæsthetist available at any hour of the day or night, or excellent operating facilities, or that partial gastrectomy in the presence of a perforation is technically easier to perform because of the œdema of the tissue planes, is no excuse *per se* to perform a gastrectomy instead of simple suture.

Consequently we shall see that partial gastrectomy will settle into its rightful place as being occasionally necessary in the treatment of a perforated ulcer, but never as a matter of routine.

Summary.

The 62 patients with a perforated peptic ulcer admitted to the Royal North Shore Hospital of Sydney from September, 1949, to June, 1953, are discussed.

Six patients were treated by aspiration therapy with three deaths, two patients received no treatment and one died, and 54 patients were treated by the conservative operation of suture with only one death.

The treatment of perforated peptic ulcer is discussed, and evidence is presented to show that the best treatment is still that of suture of the ulcer with an adequate follow-up. It is concluded that aspiration therapy is too hazardous to use because of its high mortality rate, and that emergency gastrectomy has only a limited place in the therapy of perforation.

Acknowledgement.

I wish to thank the General Medical Superintendent of the Royal North Shore Hospital, Dr. Wallace Freeborn, for permission to use the records for this article.

References.

EDITORIAL (1950), "After Gastrectomy", *Lancet*, 2: 373.
GILMOUR, J. (1953), "Prognosis and Treatment in Acute Perforated Peptic Ulcer", *Lancet*, 1: 870.
ILLINGWORTH, C., SCOTT, L., and JAMIESON, R. (1946), "Prognosis after Perforated Peptic Ulcer", *Brit. M. J.*, 1: 787.
LOWDON, A. (1952), "The Treatment of Acute Perforated Peptic Ulcer by Partial Primary Gastrectomy", *Lancet*, 1: 1270.
MORONEY, J. (1953), "Colonic Replacement and Restoration of the Human Stomach", *Ann. Roy. Coll. Surgeons England*, 1: 328.
NUBOER, J. (1951), "Primary Partial Gastrectomy for Perforated Ulcer", *Lancet*, 2: 952.
ROSE, T. (1950), "Perforated Peptic Ulcer: The Mortality and Morbidity of Treatment", M. J. AUSTRALIA, 1: 421.

THE LOCAL TREATMENT OF BURNS.

By DAVID L. DEY,

Honorary Assistant Surgeon, the Royal Alexandra Hospital for Children; Honorary Assistant Surgeon to the Plastic Surgery Unit, the Royal North Shore Hospital of Sydney, Sydney.

BURNS of varying extent are among the most common lesions in patients admitted to surgical wards, but in spite of this there can be little doubt that their treatment is capable of improvement. Clarkson and Lawrie (1946) state that the following periods are required for complete healing: (i) twenty to thirty-five days for areas up to 2500 square centimetres (roughly 15% of the body surface); (ii) forty to sixty days for areas from 2500 to 5000 square centimetres. Complete healing is rarely achieved in these times, particularly in the second group. Examination of records has shown that periods of ninety days have been not uncommon, and, now that improved resuscitation saves more lives among the severely burned, such patients may be a severe drain on both the available beds and the hospital facilities. It is of significance that Clarkson and Lawrie have been careful to point out that the longer period taken in the second group is a result of the poorer general condition of the patients, and not directly of the larger area burnt.

The complications of a burn, in the order of their appearance, are as follows: (i) shock, (ii) sepsis, (iii) scarring. To the second of these is allied the gross negative nitrogen balance resulting from any considerable breach of the surface epithelium. Such a negative balance, due to loss from the raw surface, is potentiated and increased by the presence of infection. However, even in a case free from gross infection, should the area exceed a critical level of something less than 10% of the body surface, there follow after three to four weeks inevitable emaciation and loss of morale. The dramatic change in the patient's condition, and in his outlook on life, which follows a successful plugging of the leak by grafting is one of the most satisfactory surgical rewards.

Shock kills in the first few days. Subsequent deaths are from toxæmia and nitrogen loss. Shock is a product of the thermal injury. Sepsis and scarring are the result of the breach in the epithelial covering of the body. Shock is treated by replacement of the lost circulating blood. Sepsis and scarring are prevented, or at least minimized, by replacement of the lost surface epithelium. The achievement of this at the earliest possible moment must be the whole aim of local treatment. Any method in which this is not the primary object should be discarded.

Sepsis has two local evil results: (i) it converts a "superficial" burn into a "deep" burn (*vide infra*); (ii) it leads to increased production of granulation tissue, hence to increase in subsequent fibrous tissue formation, and therefore to greater loss of function. No matter what progress is made in the control of existing sepsis, and no matter what precautions are taken against reinfection, the patient is never safe until the continuity of the surface epithelium is restored. When natural healing will not occur in three weeks or less (in the absence of infection), epithelial "cover" must be provided by skin grafting.

This conception does away with the need for complicated classifications, such as that of Dupuytren. Burn cases fall into two classes only—those which need grafting and those which do not: deep burns and superficial burns; burns in which skin loss is complete and those in which it is incomplete.

DIAGNOSIS.

The actual mechanism of the production of the burn—moist heat, dry heat, electricity *et cetera*—is of little importance beyond giving an indication of the probable depth. Thus boiling water spilt on exposed skin is likely to produce a superficial burn; if it is held in contact with the skin by clothing, a deep burn. More than momentary contact with dry heat, such as may occur when clothing catches fire, or with strong acids or akalis, is likely to produce deep burns, and electricity and splashes of molten metal will certainly do so. Large burns will naturally tend to vary in their depth in various parts.

The diagnosis of the depth of a particular recent burn may be a matter of supreme difficulty. Various methods of distinguishing the deep areas have been advocated, such as pricking with a sterile cutting needle to test for pain and bleeding. All these methods are fallacious because of progressive capillary changes, producing thrombosis and loss of tissue, which occur over twenty-four to forty-eight hours. They are better neglected.

At the end of twelve to eighteen days the areas of slough have declared themselves and the appearances at this time will determine further treatment. It suffices in the initial stages to group the cases as follows: (i) small burns: (*a*) superficial, (*b*) deep; (ii) large burns: say greater than six inches square. The actual dividing line between a small and a large burn is, of course, indefinite, but the majority of burns belong clearly to one or other group. Small burns are not complicated by shock, but a proportion of patients in the second group suffer from shock, and its treatment must take precedence for a few hours at least.

FIRST AID.

All burns are virtually sterile immediately after their receipt. The heat which coagulates or destroys the epithelial cells also coagulates the bacteria. Subsequent infection in the majority of cases is from the noses and throats of the patient or his attendants, and adequate masking at all times will avoid this.

Protection of the burnt surface with a sterile cloth (or failing this some freshly laundered linen) prevents contamination and helps a little with the pain. This is all that is necessary to be applied locally prior to definitive treatment, and the application of butter, flour, carron oil *et cetera* is to be deprecated, as these substances must be removed subsequently with considerable pain to the patient and trouble to the surgeon.

DEFINITIVE TREATMENT.

The Small Superficial Burn.

The very small burn of the household type rarely reaches the medical practitioner and is treated quite efficiently in the home by the application of "Tannafax" or some form of occlusive dressing, after a preliminary washing with soap and water.

In the treatment of a somewhat larger burn much the best way is to proceed along the lines laid down below for the "closed" method. The dressings are left *in situ* for some ten to fourteen days, and at the end of this period are removed from the healed surface. This régime saves the patient much pain and avoids the risk of introducing infection and delaying healing, even if it does appear a very passive attitude. Daily dressings are fraught with danger.

The Small Deep Burn.

The small deep burn illustrates the ideal of treatment for complete skin loss. From its production the burn undoubtedly involves the full thickness of the skin, but at the same time is not of formidable extent. The indicated treatment is immediate excision of the burnt area and closure of the wound, in a few cases directly by suture, but usually by the application of a split-skin graft. As a result healing is complete in at most fourteen days, and the saving in pain and time is paralleled by the preservation of function. With the protection afforded by antibiotics this method can be practised successfully even several days after the receipt of the injury, in fact up to the time when infection becomes established. This is the accepted doctrine of primary closure of all areas of skin loss, no matter what their mode of production, evolved in the recent war, and brought to fruition with the introduction of penicillin.

In actual fact this principle is applied to the large deep burn also, but here it must be modified to allow for recovery from shock, for difficulties due to the size of the area involved, and for the impossibility of distinguishing certainly the areas of complete and incomplete skin loss in a recent burn.

The Large Burn.

Beyond the application of a sterile cover, treatment of the large burn is delayed until shock, if present, has been controlled by vigorous fluid replacement therapy. It may then proceed along one of two lines, which appear at first sight to be diametrically opposed, but which are actually closely related to one another, as suggested by Wallace (1949). Both should aim at drying the burnt surface, and hence making conditions less suitable for bacterial growth. They may be designated as (i) the closed method and (ii) the open method. In both methods a "blanket" control of infection by means of the parenteral administration of antibiotics is of first importance.

The Closed Method.

Both lines of treatment have been employed over the years, but recently the closed method has been more generally used. It has the advantage that at the dressings the burn can be seen clearly. It is reasonably comfortable for the patient, but requires a good deal of attention to detail. Its chief disadvantage is the large amount of material necessary. It is difficult to apply in relation to the perineum, to the face, and to the necks of children, because of soiling with food or excreta, or because of movement.

Step 1.—With full aseptic ritual the burnt surface is washed over with a detergent antiseptic, such as "Cetavlon", to remove dirt, grease and other foreign material. The cleansing should not be unduly vigorous, but induction of light general anæsthesia may be a humane procedure when the burns are extensive. Morphine or procaine given intravenously may provide sufficient analgesia. The aim is to deal with gross contamination. Obviously dead loose skin is snipped away. Blisters are pricked and the serum is allowed to escape, so that the raised epithelium settles back onto the dermis.

Step 2.—A layer of *tulle gras* is applied, followed by several layers of gauze, and then a voluminous covering of cotton-wool or other absorbent material. The whole is finally compressed by means of crêpe bandages, over which may be placed a light plaster of Paris cast. If a limb is involved, this is elevated, preferably by suspension from an overhead beam. The voluminous absorbent layer has two objects: (i) to soak up serous exudate as it forms, and hence to keep the burnt surface dry; (ii) to prevent the outer surface from becoming wet and hence to maintain the sterility of the burn. Once soaking of the dressing extends to the surface, many organisms can penetrate even a very thick dressing with great speed. At all times close attention must be paid to this point and reinforcing pads applied as necessary. Besides this, the size of the packing helps to immobilize the burnt areas, while the compression and elevation limit œdema in the tissues and exudate from the surface.

Step 3.—Step 3 follows after about ten days, when the burn is dressed in the operating theatre, "Pentothal" anæsthesia being used if it is extensive. At this time a fairly accurate estimate can be made of the future necessity for grafting. A similar dressing is applied after the inspection. Should there be no areas of deep loss, a further seven to ten days will suffice for the completion of healing. Areas of deep loss necessitate the next step, and warning of this may have been given by continued gross serous loss, soaking the dressing and requiring attention at an earlier period than ten days.

Step 4.—After a further seven days the patient is again anæsthetized, the dressings are removed, all areas of slough are excised cleanly, and the whole of the raw surface is covered by means of thin split-skin grafts cut from the unburnt areas. At this time the sloughs are still firmly adherent and are removed by sharp dissection. There is considerable blood loss, and the procedure tends to be a long one, not infrequently associated with an appreciable degree of shock. Hence it is wise, when the burns are of more than minor degree, to set up a drip transfusion of whole blood before the operation commences. Since anæmia commonly appears in the more extensively burnt patients at about this time, the transfusion may be made to serve a double purpose.

The skin is best applied in the form of patches, in size roughly two inches by one inch, cut after the thin graft has been spread on *tulle gras* for ease of handling. These are placed with only narrow gaps between; but even so these gaps increase considerably the area which can be covered with a given piece of skin. Furthermore, they are much more certain in their "take" than are large sheets of skin, apparently because they do not tend to be floated off by accumulation of discharge or exudate from the underlying raw surface. The ultimate cosmetic effect is reasonably good, but in this regard it should be remembered that the main object at this time is simple epithelial cover. Any excess skin cut is refrigerated for application at the next dressing to any areas of failure.

In a small proportion of cases it may be difficult to be certain at this dressing that sloughs represent full thickness loss. Should the raising of one corner of the slough for a short distance fail to settle the question, it is better to wait a few more days rather than to risk excision of any considerable area of incomplete skin loss. The cosmetic and functional result of the natural healing of such an area is unquestionably much superior to the usual results of grafting, however successful.

Step 5.—In a further week's time the grafts are dressed and remaining raw areas, if they exist, receive further grafts. This process is repeated at similar intervals until healing will obviously follow within a matter of days. The narrow strips left between the patches disappear rapidly.

It is possible to use the same donor areas a second time after a few weeks if the previous grafts were cut very thin.

Comment.—It will be apparent that this routine requires the expenditure of time and interest on the part of the surgeon; supervision of the whole treatment must be a personal affair, and this is dictated by the active approach, which contrasts sharply with "leaving it to Sister to clean up a bit first". The saving in ward work will compensate the staff for the smells which tend to surround these patients.

The Open Method.

The open method is first cousin to the idea of "tanning" and has been developed in recent years because of the fear of mass burn casualties from an atomic explosion. In such circumstances it has the virtues that it is simple in application and that it does not call for large amounts of material or large numbers of attendants. These virtues also have appeal in ordinary practice. Its success is dependent upon the antibiotics, which are used parenterally in adequate doses. However, it should not be forgotten that this method too is only another form of preparation for the early grafting of deep burns.

Step 1.—Step 1 consists of a preliminary cleansing, which is similar to that described in the closed method. However, in this case the loose epithelium over blisters is removed. Thereafter the burnt area is left exposed in the ward. Beyond sterile towels covering adjacent blankets, and immobilization and suspension when these are possible, no special precautions are observed. No masks are used except for periods of personal attention, and the ward is swept in the usual way. The surface dries rapidly and a smooth crust is formed in some twenty-four hours, although further drying continues for some days. Longer periods indicate deep burns. After forty-eight to seventy-two hours the crust is tough and resistant. Spraying of the burn with penicillin or penicillin-lactose powder appears unnecessary and, indeed, creates "mess" from caking of excess powder.

Step 2.—In cases of incomplete skin loss the crust so formed begins to peel off after some two weeks, an epithelialized surface being uncovered. The process is precisely similar to the ordinary healing of a superficial abrasion under a scab. In the interval the patient is comfortable and remarkably free from apparent reaction. Should this process appear delayed and separation not occur at this time, complete skin loss is to be suspected and a further active phase entered.

Step 3.—The patient is taken to the operating theatre at the end of eighteen days and a light anæsthetic given. The remaining adherent crust is peeled off, and if the surface beneath is healed, or nearly healed, so much the better. Failure to peel readily denotes a slough, which is removed by sharp dissection, and the raw area is grafted exactly as has been described above in the previous section. From this point the two procedures run along parallel lines.

It is to be stressed that undue delay in instituting excision of adherent slough is dangerous. The margins of the crust lift as a plane of separation between slough and living tissue is established, exposing this moist area to bacterial invasion. Once this occurs, grafting becomes much less certain, valuable time is lost, and residual scarring is increased. The time-table is a fixed one, as opportunity for grafting in an uninfected field is fleeting.

Infected Burns.

The appearance of infection does not relieve the surgeon of his responsibility in securing early skin cover. In fact, it means that he must redouble his efforts, as each successful graft reduces the size of the problem and speeds the solution. The aim is to produce a flat, pink, painless, granulating surface, not bleeding easily, and with a minimal discharge. Such appearances are more important in determining the time for grafting than are reports from the bacteriologist. On such a surface thin split-skin grafts will "take" with a regularity approaching that seen in the case of fresh surgically prepared surfaces.

In securing such a state the following measures are helpful: (i) the daily use of "Lux" soap baths; (ii) the use of dressings moistened with 1 in 1000 eusol solution in the case of sloughing surfaces; (iii) the application of moist dressings of boracic solution (1 in 60), particularly in infections with *Pseudomonas pyocyanea*. This must be used with caution in larger areas because of the risks of absorption.

However, the slavish awaiting of ideal conditions is unnecessary. Thin postage-stamp grafts will often "take" under apparently unfavourable conditions and greatly speed control of the situation. The attempt should be made as soon as the granulations do not bleed very readily and as soon as excessive discharge is reduced. A satisfactory surface is often obtained if infected sloughs are stripped off and an absorbent dressing is applied for five or six days. This is not infrequently the course imposed in the treatment of burns not seen *de novo*, which have been the subject of frequent inspection.

Should raised œdematous granulations persist in spite of dressings, perhaps combined with the use of "Chloromycetin" (5%) in propylene glycol as a local application (suggested by Flint, Gillies, and Reid in 1952), the granulations may be stripped down to their fibrous base, bleeding controlled by pressure with hot packs, and the thin grafts applied in the usual way.

Exposure and drying of infected burns has been advocated by Wallace, but an admittedly small personal experience of this has been disappointing.

It scarcely needs to be added to the foregoing that the general condition of the patient must be closely watched. A high protein, high vitamin intake is maintained as far as possible, and anæmia is treated as it occurs by means of blood transfusion.

Discussion.

No claim to originality is made for the views expressed above; but personal experience of these methods has amply justified the claims of other authors. In any discussion of the treatment of burns one is accustomed to hear much talk of the value of various local applications. The fact is that the only dressing for a burn involving complete skin loss is a skin graft cut from the patient's unburnt skin.

The late treatment of scarring is beyond the scope of this paper.

Summary.

1. The necessity for early skin cover in the treatment of burns is indicated.

2. The means of obtaining this early cover are discussed, with consideration of two alternative courses of action and some short indication of the procedure adopted when such measures fail and infection occurs.

References.

CLARKSON, P., and LAWRIE, R. S. (1946), "The Management and Surgical Resurfacing of Serious Burns", *Brit. J. Surg.*, 33: 311.

FLINT, M. H., GILLIES, H., and REID, D. A. C. (1952), "The Local Use of Chloramphenicol in Wound Infections", *Lancet*, 1: 541.

WALLACE, A. B. (1949), "Treatment of Burns—A Return to Basic Principles", *Brit. J. Plastic Surg.*, 1: 232.

EPIDEMICS OF POLIOMYELITIS IN NEW SOUTH WALES.

By H. O. LANCASTER,

School of Public Health and Tropical Medicine, Sydney.

ALTHOUGH in general control methods against poliomyelitis have failed, there are two important preventive measures—the suspension of all tonsillectomies and the suspension of prophylactic inoculation campaigns—that should be applied when the epidemic incidence is high. We may accept the evidence that tonsillectomy is a predisposing factor to bulbar paralysis, and that inoculation of prophylactic vaccines is a predisposing factor to localized paralysis. The problem for the administrator is when to declare that an epidemic is of such an intensity as to justify the suspension of tonsillectomies and inoculations. As a criterion for the existence of an epidemic, various tests have been proposed, such as a 50% rise above the basal, non-epidemic weekly rate. In New South Wales this basal rate appears to be less than two cases per week, so that the application of the rule would be quite impractical, since an epidemic would be declared every time there were as many as three

notified cases in a week. The test would be over-sensitive and would tend to discredit control measures.

An alternative is to examine the past experience of New South Wales to determine what criterion would have led to the recognition of the major epidemics in time for control measures to be of value, and yet which would not have been too sensitive, for it is clear that the aim must be to *make a compromise between conflicting needs.* I have had access to the weekly notification figures through the courtesy of Dr. H. G. Wallace, Director-General of Health, New South Wales, and have had them plotted in a graph.

I have considered the whole State of New South Wales as the unit. It may be argued that poliomyelitis can flourish in one part of the State and not in another. If this objection was valid, then one might use the test for the metropolitan area only and let individual units in the rest *of the State declare their own epidemics. However, in* Victorian experience the first cases in an epidemic in a country area are often inoculation paralysis or bulbar paralysis, so a State-wide declaration is wise. On the other hand, it seems undesirable to hold up inoculations and *tonsillectomies indefinitely, so a provisional break from*

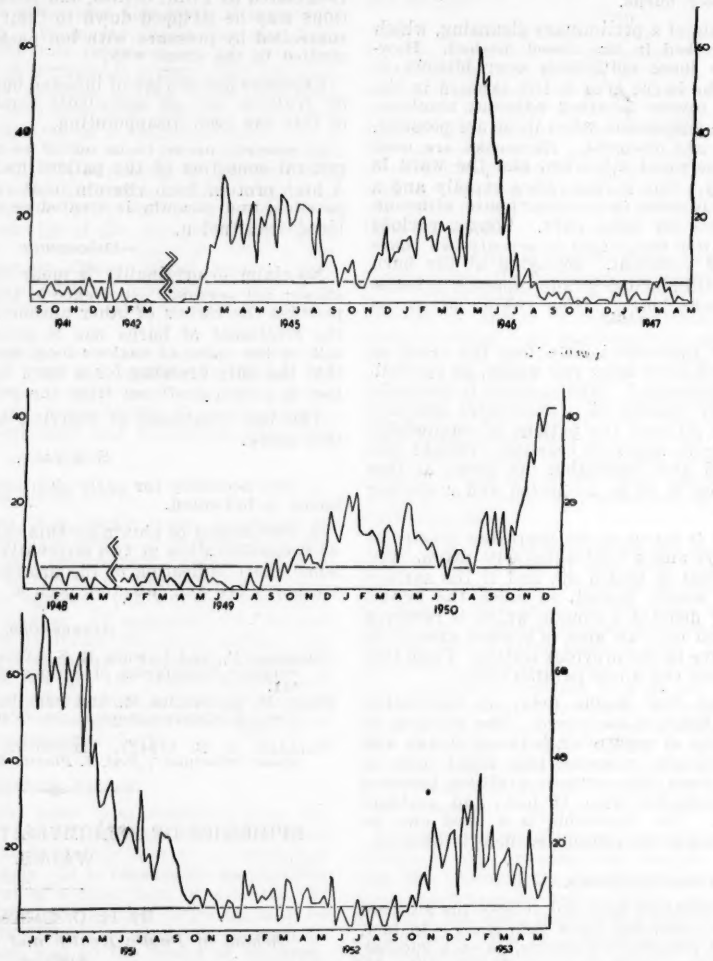

FIGURE I.

The weekly notifications of poliomyelitis in New South Wales.

Where the weekly figures have fallen below two for any prolonged period opportunity has been taken to shorten the graph by leaving the period out, but to suggest this on the graph by means of a broken line. On inspection of the graph it is evident that a useful criterion would have been the occurrence of more than five notifications in each of three successive weeks. A criterion for the end of the epidemic similarly might well have been the occurrence of less than five notifications in each of three successive weeks. Thus an epidemic would have been declared in the beginning of February, 1945, and would have been declared to be over in August, 1946. The next epidemic for administrative purposes might have been from October, 1949, to January, 1952. A further epidemic period would begin in November, 1952, and continue through to the end of the experience graphed—that is, at least to March, 1953.

the restrictions may have been permitted in October, 1951, up to October, 1952.

It appears that no criterion can be used too rigidly, as there must be a balancing between different dangers—for example, between the risk of diphtheria and the risk of inoculation poliomyelitis. The criterion presented here has the advantage of simplicity.

Acknowledgements.

This paper is published with the approval of the Director-General of Health, Canberra. The subject matter was discussed at a meeting of the Poliomyelitis Subcommittee of the National Health and Medical Research Council of Australia at the meeting in Sydney in September, 1953. Miss M. Concepción has drawn the figure.

A NOTE ON DECREASE IN AVIDITY OF THE COOMBS REACTION BY GRADUAL ELUTION OF Rh ANTIBODIES FROM THE SENSITIZED CELLS.[1]

By JEAN BARRIE, A.M.T.C., and VERA I. KRIEGER, D.Sc.,
Department of Pathology, The Women's Hospital, Melbourne.

A PECULIAR effect was observed during the demonstration of the direct Coombs test to medical students. After the red blood cells from three erythroblastotic infants had been washed three times with saline in the usual way, each final suspension gave an avid reaction with Coombs reagent when tested immediately after the last washing. The washed cells were allowed to remain at room temperature for several hours and were then placed in the refrigerator overnight. On the following day, however, it was found that the cells which were rewashed, showed only a trace of reaction with Coombs reagent in two cases, and clumping was entirely absent in the third case. In each instance avid reactions were again obtained with cells taken from the original clot and freshly washed.

This phenomenon was also noticed by a pathologist in private practice, who obtained a positive reaction to the direct Coombs test when freshly washed cells were used. The same washed cells were retested two days later, but failed to react. He referred the original sample of clotted blood to us. We found that new cells taken from this clot and tested after being freshly washed again gave a positive reaction.

In the light of these observations with the direct Coombs test, it was thought advisable to investigate whether similar effects might be involved in the indirect Coombs test. In this test unexpected results had occasionally been obtained. It was thought that the titre of a serum obtained by this method might vary in relation to the time elapsing beween the washing and testing of the cells.

Sera with titres of varying degree, but each giving avid reactions with the cells treated with the undiluted serum, were tested immediately after the third washing of the cell suspensions. They were retested after being allowed to stand at room temperature for four hours, and no apparent change in avidity of reaction or titre occurred. However, after being left in the refrigerator overnight, one specimen of serum, which gave a titre of 32 with avid reactions on the one day, showed only weak clumping and a titre of eight on the second day. Another specimen of serum originally showing a titre of three gave no reaction with Coombs reagent, even with the cells treated with the undiluted serum.

To test whether the antibodies had been eluted or destroyed, a saline suspension of sensitized washed cells was allowed to stand at room temperature for twenty-four hours. The supernatant saline was mixed with bovine albumin and fresh Rh-positive cells. An indirect Coombs test was carried out, and the positive reaction proved that the antibody had been eluted into the saline and readsorbed onto fresh Rh-positive cells.

These findings explain some peculiar results obtained on several other occasions. Cells which had been treated with high dilutions of sera whose titre was being estimated by the indirect Coombs method, and which had given definite reactions immediately after being washed, failed to react on the following day after being kept in the refrigerator overnight.

Discussion.

This observation of elution of antibodies is not the first report regarding the phenomenon. A modification of the Landsteiner-Miller (1925) technique for elution of antibodies adsorbed onto red blood cells either *in vivo* or *in vitro* has been used for removing Rh antibodies from such cells and is described by Race and Sanger (1950). The details of the technique are as follows. The cells are washed three times in an equal volume of saline. The saline-suspended cells are agitated continuously in a water bath at 56° C. for five minutes. The tubes are then transferred to centrifuge cups containing water at 56° C. and immediately centrifuged, and the supernatant fluid is removed as quickly as possible.

This technique has been used to retrieve Rh antibodies from the cells of babies suffering from erythroblastosis by Hill and Haberman (1944) and by Carter and Loughrey (1945). It has also been used in experiments on the adsorption of constituents in mixtures of antibodies.

Pickles (1949) has also reported unexpected negative results in the direct Coombs test. In discussing the value of the Coombs test in showing passive iso-immunization of the red blood cells of erythroblastotic infants she makes the following statement:

> the direct Coombs test has always been positive if the cells have been tested reasonably soon (i.e. up to two hours) after taking the blood. It has been negative, however in several cases when a saline suspension of the infant's cells has been sent through the post and we have found that weakly sensitized cells on storage, rapidly lose the antibody adsorbed on the cells.

Our observation that adsorbed antibodies can be gradually eluted into the saline medium if the cells and saline remain in contact for prolonged periods, even though the temperature is low, explains the effect reported by Pickles.

We have found that cells taken from a clot of the affected baby's cord blood are still sensitized for five days after delivery. This probably holds for any period until the cells become hæmolysed.

Finally these observations show that it is important to test the sensitized cells in either direct or indirect Coombs tests within a short time after the saline washing in order to avoid less avid reactions and sometimes even negative results. If specimens for the direct Coombs test cannot be handled immediately, whole blood and not saline suspensions of the infant's cells should be sent for examination.

Summary.

The work reported in this paper shows that the elution of Rh antibodies at 56° C. noted by other workers also takes place at lower temperatures, although more slowly. The practical application of this finding is pointed out.

References.

LANDSTEINER, K., and MILLER, C. P. (1925), "Serological Studies on the Blood of Primates. The Blood Groups in Anthropoid Apes", *J. Exper. Med.*, 42: 853.
RACE, R. R., and SANGER, R. (1950), "Blood Groups in Man", 170.
HABERMAN, S., and HILL, J. M. (1944), "The Clinical Significance of the Rh Factor. Its Importance in Erythroblastosis Fetalis", *Texas J. Med.*, 40: 182.
CARTER, B. B., and LOUGHREY, J. (1945), "A Method of Demonstrating Anti-Rh Agglutinins in Cases of Erythroblastosis Fetalis", *Am. J. Clin. Path.*, 15: 575.
PICKLES, M. M. (1949), "Hæmolytic Disease of the Newborn", 43.

[1] This work was made possible by a grant from the National Health and Medical Research Council of Australia.

Reports of Cases.

PERFORATION OF THE ŒSOPHAGUS AND RUPTURE OF THE AORTA DUE TO A PIECE OF FLY-WIRE: REPORT OF A CASE.

By J. H. W. BIRRELL and W. A. SYME,
Department of Pathology, University of Melbourne, and the Alfred Hospital, Melbourne.

PERFORATION of the œsophagus by a foreign body is today a well-recognized entity, even though not always recognized during life. One of the described, if fortunately uncommon, complications of this event is subsequent perforation of the aorta. It is thought that the perforation of both œsophagus and aorta by a small piece of wire from a fly-wire screen, in the circumstances to be described, more than justifies the present case record.

Clinical Record.

Robert B., a young, unmarried labourer, aged twenty-five years, was admitted to the Alfred Hospital on July 2, 1953, at 11.50 p.m. The following history was obtained. Three weeks previously, while he was eating his midday meal, which consisted mainly of sausages, he experienced, after a particular mouthful, a sudden attack of colicky pain which seemed to be in the front of the chest. This pain disappeared after about one minute. He then remained well until the evening meal, when he experienced a similar attack of pain on the ingestion of food, the pain being if anything more severe. During the following week he suffered this pain at almost every meal. The patient said that he felt that food caught half-way down his gullet, but when he could manage to swallow the food passed on into the stomach. Two weeks before his admission to hospital he noted that the pain had become more severe, more frequent and somewhat independent of food. The pain remained retrosternal in site and sharp and colicky in nature. One week before his admission to hospital the pain moved to the back, from whence it radiated round both sides of the chest. It had become much sharper in nature, lasting now for some ten minutes, but had little relation to eating. Occasionally still his food appeared to catch half-way down to his stomach. At this stage his temperature began to rise to 101° F. at night, his tongue was coated and his breath was offensive, and so his local doctor immediately referred him to the hospital. He had had no previous relevant illnesses. The family history was also not relevant.

On examination of the patient he was a well-built young man with a flushed, feverish appearance; his temperature was 102·4° F., his pulse rate was 108 per minute, his respirations numbered 20 per minute, his systolic blood pressure was 120 millimetres of mercury, and his diastolic pressure was 70 millimetres of mercury. No abnormality was detected on clinical examination of the head and neck, the heart, the lungs and the central nervous system. In the abdomen tenderness was obvious under both costal margins as well as in the epigastrium. Deep pressure in the last-named area produced pain in both shoulders.

A tentative clinical diagnosis of mediastinitis was made.

Some fifteen minutes after his admission to hospital, the patient vomited at least half a pint of bright, rather frothy blood, his hæmoglobin concentration then being 9·0 grammes *per centum*. After blood grouping and cross-typing, he was given a transfusion of two pints of blood; penicillin, 1,000,000 units every six hours, and streptomycin, 0·5 gramme per hour, were also given.

Next morning the chest and abdomen were radiologically examined, and examination by means of a barium bolus was also carried out. This last revealed deviation of both atria and œsophagus, with a dense shadow between, but no sign of a foreign body.

Blood examination after the transfusion showed that the hæmoglobin value was 12·3 grammes *per centum* and that a leucocytosis of 18,000 per cubic millimetre was present, with neutrophilia and a "shift to the left". He was examined early by the thoracic surgeon, and an œsophagoscopic examination was decided on for the following day. During July 3 his blood pressure remained steady, his pulse rate was 110 per minute, and his temperature rose from 102° to 104° F.

On July 4, early in the morning, he began to dry retch, and then vomited bright red blood; he died within five minutes despite intubation, suction and artificial respiration.

Autopsy Findings.

Autopsy was performed fifty hours after death, the pertinent findings being those in the thoracic cavity and in the upper portion of the alimentary tract.

The organs were removed in their entirety. It was noted that each pleural cavity contained about one pint of straw-coloured turbid fluid containing many flakes of fibrin. Examination of the aorta and the posterior mediastinal contents revealed dense adhesions to the vertebral column requiring more dissection for removal than is usual.

When the aorta was opened posteriorly from below, a small star-shaped laceration one centimetre in its greatest diameter was noted on the right anterior aspect of the aorta, 3·5 centimetres below the arch. Blood clot was apparent in the opening. The œsophagus was then opened from above when a large rent, some three centimetres in maximum diameter, was found in the left side of the

FIGURE I.

Photograph of the specimen from a subject with rupture of the aorta following perforation of the œsophagus by a piece of fly-wire. The actual wire may be seen in the aorta on the left of the photograph, the dotted line showing the site and shape of the wire during life.

œsophagus 10·5 centimetres below its upper extremity. This rent had necrotic lacerated edges and contained blood clot, being in direct continuity with the laceration in the aorta (Figure I). After some dissection a small piece of rusty serrated wire, with sharp ends and measuring 2·5 centimetres in length, was found lying between the two lacerations. It had a curve at the œsophageal end, the concavity of the curve being uppermost. The wire in its immediate vicinity was surrounded by infected blood clot, which itself lay in an oval-shaped indurated area of mediastinitis.

The stomach and upper half of the small intestine were filled with clotted blood. Apart from some œdema of both lungs, no abnormality was found in the remainder of the organs.

Examination of sections of the aortic wall at the site of rupture revealed necrosis of the whole wall, with diffuse infiltration of all coats by cells, polymorphonuclear leucocytes predominating.

ILLUSTRATIONS TO THE ARTICLE BY KATHLEEN CUNINGHAM, M.S., F.R.A.C.S.

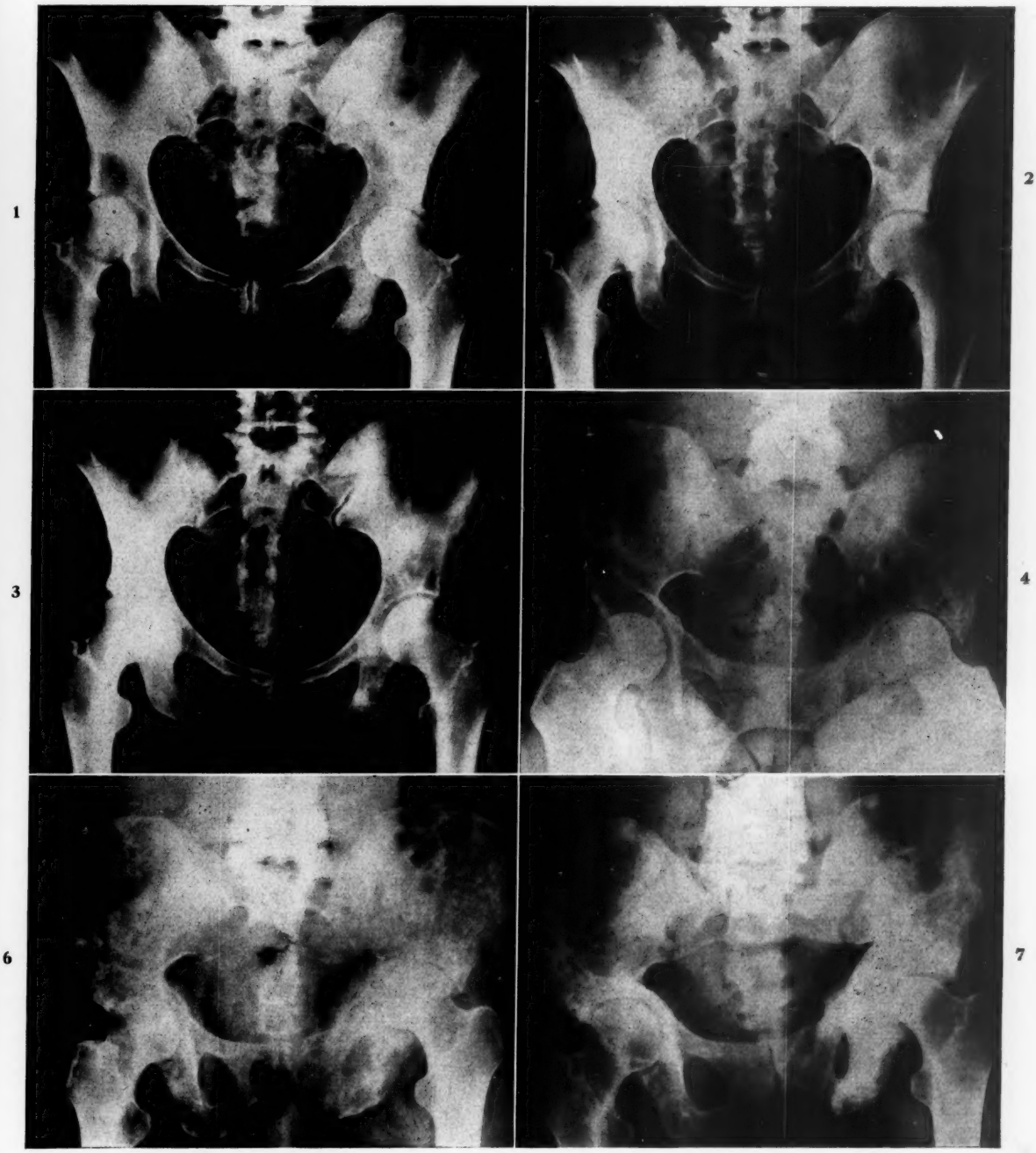

FIGURE I: Early metastatic lesions in the ilium round the acetabular cavity (June, 1952). FIGURE II: Extension of lesion into pubic ramus (December, 1952). FIGURE III: Lesion stationary (March, 1953). FIGURE IV: May, 1953: sacrum disintegrated, whole pelvis involved with metastases. FIGURE VI: August, 1953: early but definite recalcification (one month after operation). FIGURE VII: November, 1953: complete recalcification of pelvis with deformity of the pelvic inlet.

ILLUSTRATIONS TO THE ARTICLE BY JOHN FALCONER GRANT.

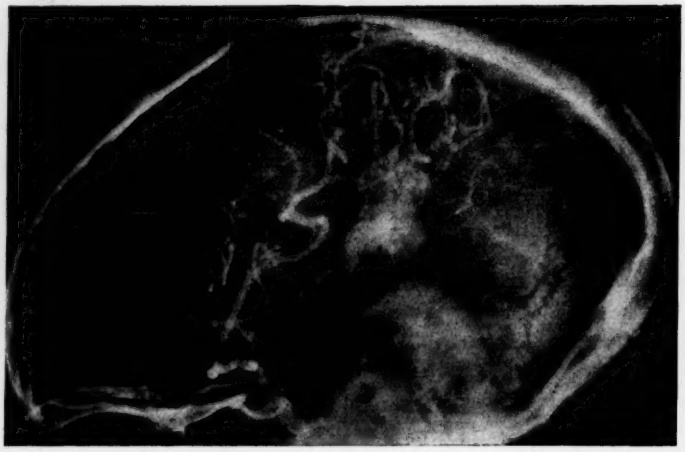

Figure I.

Figure III.

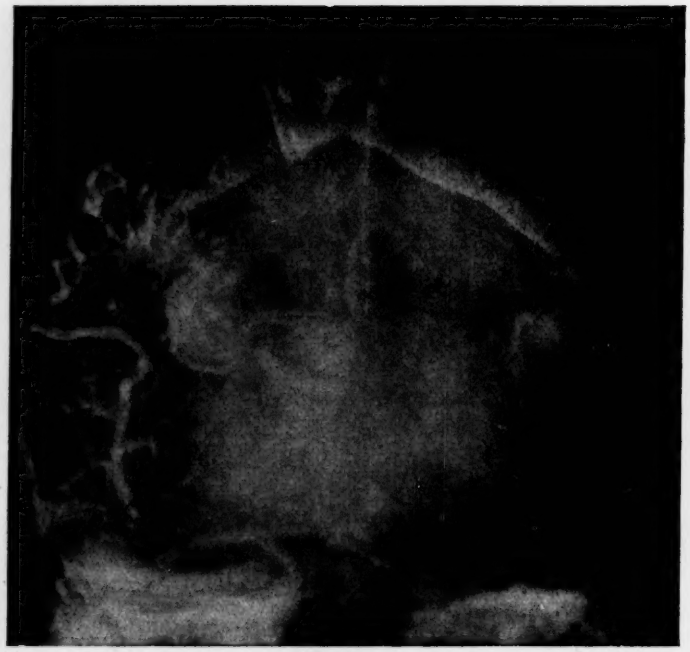

Figure II.

Figure IV.

Discussion.

Perforation of the œsophagus by a foreign body is an accident fraught with danger; this is still true today, despite the introduction of chemotherapy and the antibiotics as well as improvements in radiological and other diagnostic and therapeutic techniques.

Perforation usually occurs in the thoracic portion at one of the recognized sites of constriction, the main site being the point where the left bronchus passes across anteriorly to the œsophagus.

The perforation may be caused by the foreign body itself, by the patient's efforts to rid himself of the offending body, or very occasionally by instrumentation. Orton (1930) states that perforating foreign bodies of the œsophagus may be classified as those which perforate at once and those which perforate by erosion. Sharp fragments (as in the present case) cause immediate perforation of the œsophagus, while perforation by ulceration and erosion is usually due to prolonged sojourn of a foreign body in the œsophagus.

An extraordinary variety of objects and materials have been described as concerned in these accidents, the greatest proportion of them being pins, coins and fish-bones. Several cases have been reported in which wire (Swain, 1930) or a piece of sharp steel (Beal, 1918) perforated both œsophagus and aorta; but the circumstances surrounding the swallowing of the foreign bodies are not recorded.

In this case the foreign body was a piece of curved rusty wire 2·5 centimetres in length, which had obviously once been portion of a fly-wire screen. This wire should be recognized as a dangerous foreign body, since it is sharp and has considerable tensile strength, as anyone who has repaired a fly-wire screen will affirm. This applies to rusted fly-wire almost as much as to the new material.

Rupture of the aorta or large vessels of the neck following perforation of the œsophagus has been described by a number of authors. Thus Ter-Oganesjan (1935) was able to collect from the literature some 50 cases of hæmorrhage from the large vessels, adding five of his own.

Hæmorrhage may take place at any time from four to five days to two years (Grey Turner, 1910) after the initial perforation of the œsophagus, this hæmorrhage being seldom if ever primary. This fact is easily undersood when the structure of the large vessels is considered, since they have very thick elastic walls embedded in the looser connective tissue of the mediastinum. Hence the usual event, secondary hæmorrhage, follows the formation of an abscess in the tissue surrounding the œsophagus and large vessels.

Ulceration and rupture would, of course, be hastened or facilitated by contact of the pulsating vessel with a pointed foreign body.

In the present case impaction occurred at the classical site—namely, where the left bronchus passes anteriorly to the œsophagus. Perforation and penetration occurred almost immediately, aided, no doubt, by vigorous peristalsis. Suppuration followed with ulceration and necrosis of the aortic wall, which allowed subsequent rupture into the œsophagus.

It may be argued that the patient would have felt pain at the back of the pharynx as soon as he started to swallow. If he did experience pain on taking the mouthful of food with the wire included, the instinctive action would be to swallow harder rather than to vomit or regurgitate the object back.

In this case, of course, there was no history of actual swallowing of the foreign body, the presence of the wire being completely unsuspected. In retrospect, however, the history throughout is very suggestive.

Radiography, which is usually of great value both in making the diagnosis and in locating the foreign body, was in this case too late to allow of any effective treatment. It did reveal the area of mediastinitis with deviation of the trachea to the right; but again, even in retrospect, and with precise knowledge of the site and nature of the lesion, it is practically impossible to demonstrate the wire clearly amid the many shadows in the mediastinum. The radiological aspects, particularly in the case of a non-opaque foreign body, have been well summarized by McGibbon and Mather (1935).

While complete proof of the source from which the piece of wire was derived is not possible, suspicion attaches very strongly indeed to the sausages which the patient was eating at the time of onset of his pain.

Such an accident can conceivably occur again, and it is thought that the difficulties of diagnosis and treatment may be considerable. Early œsophagoscopic examination—that is, within two or three days of ingestion of the foreign body—may not demonstrate the fine piece of wire in the œsophagus, or even the laceration in its wall. X-ray examination of this site with such a thin foreign body is also not helpful, so that thoracotomy may need to be considered.

Summary.

1. A case of perforation of the œsophagus, mediastinitis and subsequent rupture of the aorta by a piece of wire from a fly-wire screen is described.

2. The mechanism and sequence of these events are discussed.

3. Sausages appear to have been the source of the offending foreign body.

4. The difficulties of diagnosis and treatment are mentioned.

Acknowledgements.

We desire to thank Dr. Ian McLean for permission to record this case. We are grateful to Mr. Tom O'Connor for the photograph of the specimen.

References.

BEAL, N. H. (1918), "Hæmatæmesis and Melæna Caused by a Piece of Metal from the Œsophagus Perforating the Aorta", *Brit. J. Surg.*, 5: 512.

MCGIBBON, J. E. G., and MATHER, J. H. (1935), "Perforation of the Œsophagus by Swallowed Foreign Bodies", *Lancet*, 2: 593.

ORTON, H. B. (1930), "Mediastinitis following Œsophageal Foreign Body; Report of Cases", *Arch. Otolaryng.*, 12: 635.

SWAIN, H. L. (1930), "An Unextracted Foreign Body Penetrating the Aorta of a Ten Months Old Infant", *Arch. Otolaryng.*, 12: 544.

TER-OGANESJAN, M. (1935), "Hæmorrhage due to Foreign Bodies in the Œsophagus", *Acta Otolaryng.*, 21: 116; abstracted in *J. Otolaryng.* (1935), 50: 65.

TURNER, G. G. (1910), "Death from Perforation of the Aorta by a Half-penny Impacted in the Œsophagus with Remarks on Some Similar Cases", *Lancet*, 1: 1335.

CARCINOMA OF THE BREAST WITH METASTASES TREATED BY TOTAL ADRENALECTOMY.

By KATHLEEN CUNINGHAM, M.S., F.R.A.C.S.,
Honorary Surgeon, Rachel Forster Hospital, Sydney.

Clinical Record.

MRS. X., aged forty-three years, was first examined on July 17, 1952. She had had a radical mastectomy four years before at the age of thirty-nine years for an early scirrhous carcinoma which had supervened on hormonal mastopathy. In February, 1952, she had undergone hysterectomy and bilateral oophorectomy. After this operation she complained of pain in the left leg, and an X-ray examination revealed a patch of destructive bone lesion round the left acetabulum (Figure I). Four hundred milligrammes of "Testoviron" were implanted into the rectus sheath, and she was given 100 milligrammes of "Testoviron" intramuscularly three times a week for eight weeks. In addition to this lesion she was severely thyrotoxic; she had exophthalmos and œdema round the eyelids, her basal metabolic rate was +35%, and she had tremor and tachycardia. This condition was treated by Dr. Willa Nelson with thiouracil and small doses of thyroid, and came

completely under control. The patient continued with the "Testoviron" injections, until in August, 1952, she began to have considerable œdema of the face and the front of her shins, where a condition of scleroderma developed. A urea concentration test performed at that time gave a normal result, and her blood urea content was 31 milligrammes per 100 millilitres. Her serum calcium content was 7·3 milligrammes per 100 millilitres, which is a low reading. In view of the fact that her renal function was normal the "Testoviron" injections were continued.

On August 11 the radiologist reported that some sclerosis was occurring in certain of the lesions, but others appeared to be increasing in extent. The lesion round the acetabulum definitely contained more calcium (Figure II). The patient continued with the "Testoviron" in spite of the œdema. We could not decide whether that was due to her thyrotoxic condition or to the "Testoviron" she was taking. In December the radiologist reported that the metastatic deposits had progressed to a slight extent, but that recalcification was still occurring (Figure III). The œdema increased to quite a pronounced extent, and the result of her renal function test at that time was not so good as it had been before; her blood urea content was 54 milligrammes per 100 millilitres. The serum calcium content had risen to nine milligrammes per millilitre. In spite of this poor renal function, the "Testoviron" injections were continued at regular intervals until about April, 1953. All this time the patient was ambulatory and her pain had considerably decreased. When she was not having "Testoviron" injections during the spells of three to four weeks that we gave her between injections, her pain was certainly more severe. Pronounced side-effects occurred with the "Testoviron" injections. Her voice became harsh and croaky, hair commenced to grow heavily on her legs, her clitoris was enlarged and her libido greatly increased. In April she began to develop allergic symptoms. Each time she had "Testoviron" injections her tongue and face swelled to a rather alarming extent, and I decided that as she had had so much "Testoviron" we should suspend the injections for at least a month. During that month the bony metastases progressed at an alarming rate, and on May 1 the radiologist reported as follows:

> There is further destruction in the right ilium and there is now considerable destruction of the wings of the sacrum and the appearances in the right ilium above the acetabulum are now definitely those of metastases. I think there probably is more calcium around the left acetabular cavity than when last rayed. She also has an early destructive lesion around her right acetabulum. (See Figure IV.)

Shortly after this the patient was in great pain and bed-ridden. She could not move and was under continuous sedation. She was admitted to the Rachel Forster Hospital on June 3. A blood count gave the following information: the erythrocytes numbered 4,800,000 per cubic millimetre, the hæmoglobin value was 12·9 grammes *per centum* (86%), and the blood sedimentation rate was 44 millimetres in one hour. The serum alkaline phosphatase content was 13 units *per centum*. The patient was in desperate pain, she could be nursed only with great difficulty, and required one-third of a grain of "Omnopon" almost every four hours to control her pain. Her condition was regarded as hopeless and arrangements were made to transfer her to the Home of Peace for the Dying. It was at this stage that I heard of the work of Charles Huggins and T. L.-Y. Dao, of Chicago in the United States of America (1953). He had been performing total adrenalectomy in cases of carcinoma of the breast and prostate with bony metastases with quite reasonable success in 60% of cases. I decided to give the patient a chance of having the adrenalectomy, because otherwise her condition was hopeless. She consented to undergo the operation, and on July 15 I performed a bilateral total adrenalectomy, with the implantation of 250 milligrammes of DOCA. The patient was put on a regime of cortisone, 100 milligrammes every six hours, for twenty-four hours before operation, and during operation she received another 150 milligrammes. The operation was performed in one stage. The right adrenal was removed without disturbing the twelfth rib; it was thought safer to do this in case a tear in the pleura resulted in a collapse of the right lung. After the right adrenal had been removed, the patient was turned over very slowly. The anæsthetist asked me to allow twenty minutes for turning, reposturing and getting the patient settled into the new position. On the left side, the left twelfth rib was resected and the left adrenal was removed rapidly. During operation the drip administration of saline was maintained. Her blood pressure began to fall towards the end of the removal of the left adrenal, and 30 milligrammes of "Methedrine" were given intravenously at this stage, and also an intravenous injection of atropine sulphate (1/150 grain). This resulted in a rise of systolic blood pressure to 160 millimetres of mercury and a very rapid pulse rate. (See blood pressure chart during the operation, Figure V.)

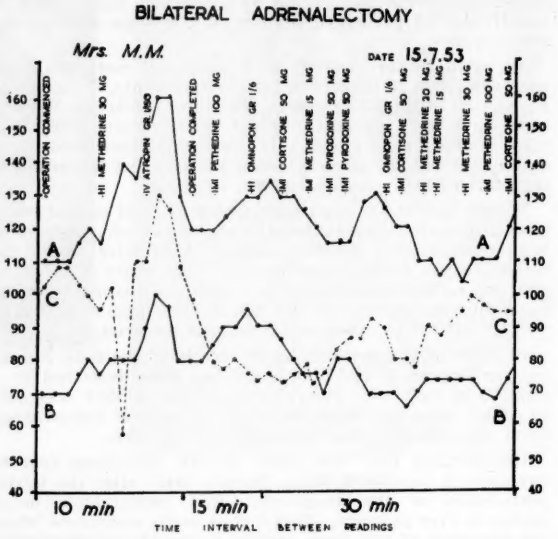

FIGURE V.

Blood pressure and pulse chart during operation and for twelve hours after bilateral adrenalectomy. A, systolic blood pressure; B, diastolic blood pressure; C, pulse rate.

The operation was completed in less than two hours and the patient was returned to bed. Her blood pressure was maintained at above 115 millimetres of mercury, systolic, and 85 millimetres, diastolic, throughout the night by occasional injections of 30 milligrammes of "Methedrine", continuous administration of oxygen and 50 milligrammes of cortisone at six-hourly intervals. Urinary output was measured by an indwelling catheter, and a careful check was kept on the fluid balance until her output was within normal limits.

She survived the post-operative period and the wounds healed well. We gradually reduced the cortisone dosage till she was taking a maintenance dose of 37·5 milligrammes a day, with two drachms of sodium chloride. Her blood calcium content before operation on June 26 was 9·26 milligrammes per 100 millilitres of blood. On June 29 a urea concentration test gave a normal result. On July 30 (after the operation) the serum alkaline phosphatase level was 9·6 units *per centum*. On August 19 the serum alkaline phosphatase level was 18·8 units *per centum*. Charles Huggins states in his article that if there is a pronounced rise in the serum alkaline phosphatase level, the outlook for the patient is very much improved. Examination of the adrenal glands gave the following information: the left adrenal weighed 7·1 grammes and the right adrenal 7·5 grammes; the histological appearances were normal, and there was no sign of any metastatic deposit.

The patient lost her pain almost immediately after the operation. She became easy to nurse, she slept well, and required only one injection of morphine or "Omnopon" daily. Her condition rapidly improved, and at the end of about five or six weeks we allowed her out of bed and got her moving on crutches and in a wheeled chair. On August 18 the radiologist reported as follows:

> Very slight improvement has taken place in the left side of the pelvis, particularly round the acetabular cavity, also in the small lesion in the fourth lumbar vertebra that had recently appeared. (See Figure VI.)

She also had a metastatic deposit in the outer end of the clavicle, but up to that date that had shown no improvement.

The patient went home on crutches and using a wheeled chair. Since her return home she has improved considerably day by day. She is now walking round the house without crutches, she takes a walk of three to four hundred yards along her street, and she is able to do light housework and take an interest in her family and her surroundings. She is happy and bright and is practically pain free except when she over-exerts herself. She is maintained on ordinary aspirin and codeine derivatives, with possibly one injection of "Omnopon" or morphine at night. She looks well, and she is eating and sleeping well.

She was radiologically examined again on November 10, and the report was as follows:

> Complete reconstruction of her pelvis has occurred with heavy deposits of calcium in all areas, especially round the left acetabulum. Some deformity of the pelvic inlet has occurred owing to the extremely spongy consistence of the pelvic girdle before recalcification. The lesion in the fourth lumbar vertebra has healed and the one in the clavicle has almost recalcified. (See Figure VII.)

Her serum chloride content is 615 milligrammes per 100 millilitres, and her serum alkaline phosphatase level is three units *per centum*.

Comment.

The rationale of total adrenalectomy is that it removes the last traces of œstrin secretion from the body. After a bilateral oophorectomy, which in my opinion should always be performed on patients who are pre-menopausal, Huggins states that there are still quite high levels of œstrin excretion in the urine. After adrenalectomy, the œstrin excretion is nil.

Very little further comment is necessary on this patient. A hopeless, moribund, pain-racked patient with extensive bony metastases in the pelvis, the fourth lumbar vertebra and the clavicle, has been converted into one who can return to her home and attend to her own needs and those of her children, and is improving daily in mental outlook and happiness. Some pain of an arthritic nature is present, probably owing to the enforced inactivity. I would allow her very little movement until after the last X-ray examination.

Although the disease appears to be arrested, the future obviously cannot be foretold. The question may arise whether this treatment will also have any effect in other forms of carcinoma in organs under the control of œstrin —for example, carcinoma of the *corpus uteri*, cervix, vagina and vulva.

The case is reported as a single example of this type of treatment, and no definite conclusions can be drawn from it. More cases will have to be observed and much time must elapse before this can be done.

Acknowledgements.

I am indebted to Dr. Colin Edwards, Honorary Urologist of the Rachel Forster Hospital, for sending me by air mail an account of his interview with Dr. Charles Huggins and of the pre-operative and post-operative régime of cortisone and DOCA. I also wish to thank Dr. Philip Jobson and Dr. Gwenifer Bernard for an extremely skilful anæsthetic, and Dr. Marjorie Dalgarno and the radiologists of the Mater Misericordiæ Hospital, North Sydney, for their interpretation of the X rays. My thanks are also due to Dr. Willa Nelson, Senior Physician of the Rachel Forster Hospital, for her help in controlling the thyrotoxicosis, and for her helpful suggestions with the blood chemistry in the post-operative period.

Reference.

HUGGINS, C., and DAO, T. L.-Y. (1953), "Adrenalectomy and Oophorectomy in Treatment of Advanced Carcinoma of Breast", *J.A.M.A.*, 152: 1388.

INTRACRANIAL ANGIOMA WITH OPERATIVE CURE: REPORT OF A CASE.

By JOHN M. F. GRANT,
Sydney.

WHILST the surgical treatment of intracranial aneurysms both by direct and by indirect attack is now a firmly established and almost everyday procedure in neurosurgical centres, the operative attack on the intracranial angiomata is still comparatively uncommon. The reason for this is obvious, as in a relatively small community these lesions remain uncommon, even though they are being diagnosed more frequently since the introduction of cerebral angiography.

The case is considered worth reporting, not only because of its rarity and the successful operation result, but because it illustrates the clinical course of the condition.

G.D., aged nineteen years, was admitted to the Royal South Sydney Hospital in November, 1952. Two weeks prior to his admission he had fallen off his bicycle and had suffered a mild concussion. He recovered from this episode and was well until one day prior to his admission to hospital, when he developed a headache. This was severe enough to prevent him from playing more than two games of tennis that night; however, he slept well, but next morning he still had a mild frontal headache. About midday the headache became more generalized and very intense. He became confused and was transferred to hospital.

His previous history was interesting. Some ten years before this present illness he had suffered an episode of left hemiplegia following a fall and minor head injury. He had been admitted to hospital at that time and stated that he had been operated on for a "depressed fracture of the right temporal region". He had recovered from this illness and had suffered from no disability whatsoever.

He had never suffered from headaches or fits.

On admission to hospital he was confused and disorientated; he vomited and had marked meningismus with photophobia. He had a complete left hemiplegia with some blunting of sensation on the left side. There was mild congestion of the retinal vessels. A curved scar in the right temporal region could be seen, but no evidence of any bony defect could be felt. No intracranial bruit could be heard, and the absence of this was confirmed later.

The findings from the remainder of his clinical examination were essentially normal.

A lumbar puncture was carried out, which showed blood-stained fluid under increased pressure. X-ray examination of the skull showed no abnormality; no evidence of either recent or old fracture could be seen.

Right common carotid angiography was next carried out under "Pentothal" anæsthesia. The films, as illustrated in Figures I and II, show in lateral and antero-posterior projections the angioma in the fronto-parietal region extending deeply toward the mid-line into the brain. One large feeding vessel arising from the middle cerebral artery is well demonstrated. The typical wedge-shaped distribution of these lesions can be well seen and, in addition, a large coiled collection of dye contained in a false aneurysmal sac, which probably has resulted from intracerebral hæmorrhage ten years ago, at the time of his "post-traumatic hemiplegia".

The patient was at this stage transferred to the Royal North Shore Hospital of Sydney.

Under general anæsthesia the lesion was exposed and excised after a large osteoplastic bone flap had been turned down in the fronto-parietal region.

The pathology of these lesions makes removal possible. As the result of previous and recent bleeding an intracerebral hæmatoma is formed. The angiomatous tissue lies in the wall of the hæmatoma and hangs down into the clot like a bunch of grapes. A fibrous reaction forms around the lesion and produces a false capsule, and this defines the limit of the resection.

The patient had a stormy convalescence for the first week. An extradural hæmatoma occurred on the third day and was removed. After this he steadily improved and has now returned to his previous occupation and is again attending the technical college. He has a minimal spastic paresis, which does not interfere with his occupation and is still decreasing.

Microscopic examination of sections shows the typical angioma (Figure III); and in a section of the aneurysmal sac is a fibrous wall with organized clot and fluid blood in the centre (Figure IV).

The surgery was carried out under basal "Avertin" anæsthesia supplemented by "Pentothal", administered by Dr. Clive Paton. Hypotensive anæsthesia was not used, as the patient was a healthy young adult, and it was felt that "Avertin" would produce a sufficient fall of blood pressure to allow control of hæmorrhage.

Summary.

1. A case of intracranial angioma was treated by excision.
2. Typical radiological appearances were seen on angiography.
3. Microscopical sections showed typical appearances of the lesion.

CARDIAC ARREST, WITH RECOVERY AFTER CARDIAC MASSAGE.

By P. W. VERCO,
Adelaide.

IN recent numbers of this journal some prominence has been given to the procedure to be carried out if cardiac arrest should occur during the course of a general anæsthetic. Sadove, Wyant and Gittelson, in an excellent review of the management of cardiac arrest, have directed attention to the cause of death after the heart has been successfully restarted. These authors reported striking alleviation of coma in such a case following the intravenous injection of hypertonic glucose solution.

Clinical Record.

On the morning of August 21, 1953, percutaneous carotid arteriography was carried out on a housewife, aged forty-eight years, who, during the past five years, had suffered from repeated attacks of *petit mal* affecting the jaw and left hand, and from several attacks of *grand mal*. An electroencephalogram had suggested a lesion in the right hemisphere. Lumbar puncture had disclosed that the cerebro-spinal fluid was under a pressure of 100 millimetres, but contained no abnormal amount of cells or protein.

Thiopentone was given intravenously and during the course of the examination the patient became deeply cyanosed and respiration ceased as the tongue was being pulled forward to improve the airway. A moderate hæmatoma was present in the neck. The apex beat was sought immediately and as no cardiac pulsation could be felt, artificial respiration was carried out whilst a scalpel was brought. An incision was then made in the fourth left anterior intercostal space; the heart was found to be flabby and in diastole. Cardiac massage was commenced, the heart being compressed as frequently as possible against the sternum.

No accurate time check was kept, but it was estimated that artificial respiration was carried out for half a minute and that cardiac massage was commenced within a minute of cardiac arrest. The heart, after several abortive contractions, commenced to beat some half a minute later.

Very fortunately, expert assistance was available in the hospital, and a thoracic surgeon and anæsthetists appeared on the scene as if by magic.

Oxygen was administered into the hypopharynx, as some difficulty was experienced in intubating the larynx. Finally, a small bronchoscope was passed and oxygen was administered through it under pressure. Each time intubation of the larynx was attempted and the airway was obstructed temporarily it was remarkable how feeble the cardiac pulsation became. The wound in the thorax was then blocked by hand as much as possible, whilst oxygen was administered by the endotracheal route for a time. The force of the heart's action improved noticeably and the wound was sutured in layers an hour after the thoracotomy, when the force of the heart's contraction and the pulse at the wrist showed no further improvement. The blood pressure at this stage was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic, and the pulse rate was 80 per minute. A drain had been inserted through a stab wound in the fifth left intercostal space in the axillary region, and attached to an underwater seal. A film of the chest revealed a small left pneumothorax. At this juncture the bronchoscope was removed, a rubber endotracheal tube was passed, and a stomach tube was passed through the nose. A laceration of the posterior pharyngeal wall was sutured, and the patient was returned to her room, oxygen being administered continuously.

The patient remained in deep coma until about five hours after the incident, when she roused a little, opened her eyes, and turned her head. She soon lapsed into coma again and, two hours later, two general epileptic seizures occurred.

A lumbar puncture was performed and the cerebro-spinal fluid pressure was found to be 250 millimetres by manometer. This was reduced to 150 millimetres by draining off five cubic centimetres of fluid. The fluid was clear and did not contain an abnormal amount of cells or protein. Before the lumbar puncture the patient was deeply comatose and would not respond to any painful stimuli, even the lumbar puncture. A small magnesium sulphate enema was given. A quarter of an hour later there was a pronounced lessening of the coma and the patient opened her eyes and moved both arms and legs spontaneously, and the enema was returned. This improvement was maintained for a futher hour. She then became comatose again, but roused a little during the night, attempting to pull out the endotracheal tube, so it was removed.

Next morning the lumbar puncture was repeated and the pressure of the fluid was found to be 170 millimetres; this was reduced to 100 millimetres, the fluid still being clear and colourless. Again there was a striking improvement in the level of consciousness, the patient roused sufficiently to obey commands, move all her limbs, and complain about her very sore pharynx. Further improvement occurred during the day and feeding was commenced via the stomach tube at noon.

The intramuscular administration of penicillin had been commenced early, 2,000,000 units being given every four hours. Sedatives had been withheld, because it was not wished to give any further respiratory depressant. On the second day it was necessary to give pethidine for pain in the chest and headache consequent upon her sitting up after lumbar puncture. The wound in the pharynx was oozing a little blood

On the third day the pulse rate increased and it became irregular. An electrocardiogram revealed paroxysmal ventricular tachycardia. This irregularity was readily controlled by the oral administration of quinidine and the blood pressure rose to 140 millimetres of mercury, systolic, and 80 millimetres, diastolic. Signs of consolidation were

apparent at the base of the left lung, an X-ray film of the chest revealed a very small effusion, and the temperature became elevated. The pharynx had healed, feeding was commenced by mouth, and the intercostal drain was removed. The patient's condition continued to improve, though she was a little disorientated and easily fatigued mentally.

On the fifth day her condition had improved still more, despite the fact that her temperature had risen to 103° F. The pulse was regular and of good volume. She complained of some pain beneath the right breast as well as in the region of the wound. The chest was aspirated and some four ounces of clear brown fluid were removed from the left pleural cavity. The patient insisted on using a commode and it was necessary to give sodium phenobarbital in frequent doses by intramuscular injection to keep her in bed. X-ray films of the chest revealed some consolidation in the middle lobe of the right lung, as well as in the lower lobe of the left lung. Despite the continued administration of penicillin in massive doses, the pyrexia had increased, so terramycin was given as well. The temperature settled by lysis over the next five days, as clinical evidence of consolidation disappeared.

The patient's mental and physical state steadily improved and she was discharged from hospital on the sixteenth day. Several attacks of *petit mal* occurred in the second week and the patient's mental faculties had returned to normal by the end of the second week.

Discussion.

Interest in resuscitation after cardiac arrest has been stimulated by letters in the correspondence portion of this journal.

Since this experience my attention has been directed to an article by Sadove, Wyant and Gittelson. Their patient was comatose after resuscitation by cardiac massage and striking alleviation of the coma was secured by the intravenous administration of 50 cubic centimetres of 50% glucose solution. This alleviation lasted for half an hour, when coma deepened but did not attain the previous level. This sequence was repeated after four hours and then one litre of 25% glucose solution was given by the slow intravenous drip method with gradual lightening of the coma. Their patient regained consciousness on the third postoperative day, and thereafter made an uneventful, although slow, recovery, with no residual neurological symptoms and no evidence of kidney damage when discharged from hospital ten days later.

Patients who have been resuscitated by cardiac massage may remain comatose, or death may be ushered in by a series of convulsive seizures. Those who recover may show considerable impairment of cerebration. In this instance it was reasoned that the cerebral complications were likely to be due to œdema following the period of oxygen deprivation. It was deemed that the risk of a medullary or tentorial cone was negligible in view of the long history and the absence of papilloedema, so lumbar puncture was performed in preference to the intravenous administration of hypertonic glucose solution.

This case is reported to draw attention to two points. First, when faced with respiratory and cardiac arrest, it is possible for those without surgical skill to commence artificial respiration and, when this fails, to open the thorax and provide rapid manual compression of the ventricles whilst oxygen is being administered. A very short time only is available after cardiac arrest occurs to restore cerebral circulation; this is probably about ninety seconds (Burnell). Any aseptic precautions are too time-consuming to be considered. Should the heart be started, the assistance of one's surgical colleagues can be obtained to close the wound.

Secondly, attention is drawn to the concept that, after the anoxia, cerebral œdema develops and may respond dramatically to controlled spinal drainage coupled with dehydration therapy, as in this case, or to the intravenous administration of hypertonic glucose solution.

Acknowledgements.

My thanks are due to Dr. Mary Burnell for stimulating my interest in this method of resuscitation, and my gratitude to Dr. J. Rice, Dr. J. Stace and Dr. H. D. Sutherland for help given so unstintedly in an emergency, and to Dr. R. F. West, Dr. N. R. Bennett and Dr. T. A. R. Dinning for their assistance and advice.

Reference.

SADOVE, M. S., WYANT, G. M., and GITTELSON, L. A. (1953), "The Acute Hypoxic Episode", *Brit. M. J.*, 2: 255.

A CASE OF EXTENSIVE PERFORATION OF DUODENAL ULCER TREATED BY IMMEDIATE GASTRECTOMY.

By W. H. NEILD, B.A., M.B., M.S.,
Newcastle.

Clinical Record.

MR. X, aged fifty years, an oysterman, was admitted to the Royal Newcastle Hospital on November 23, 1949. He had had a duodenal ulcer (proven by radiography) for the previous ten years, and over the past few months had suffered very severely from ulcer pain. In the previous month he had entered hospital with the object of submitting to partial gastrectomy. However, private business had necessitated his discharge. On November 23, at about 10 a.m., there had been an acute exacerbation of his ulcer pain, and his family doctor had requested his admission to hospital. When the journey was almost complete (a distance of 50 miles) the pain became suddenly agonizing in the upper part of the abdomen.

Examination of the patient revealed the classical features of a ruptured ulcer. Under general anæsthesia the abdomen was opened by a right upper paramedian incision. There were about three pints of greyish free fluid in the peritoneal cavity, and when the right lobe of the liver was elevated a perforation was found in the first part of the duodenum. The perforation was oval in shape and one and a half inches in its long axis. Most of the anterior and superior aspects of the first part of the duodenum were missing, having been destroyed by the perforation. The ulcer extended onto the posterior aspect of the duodenum, where it was strongly adherent to the pancreas. Satisfactory closure could not be performed, and partial gastrectomy was undertaken forthwith. It proved difficult to secure sufficient tissue to invaginate the duodenal stump owing to the adherent posterior portion of the ulcer. However, this was achieved after a tedious dissection. Two-thirds of the stomach were removed and gastro-intestinal continuity was reestablished by antecolic iso-peristaltic anastomosis with a Finsterer valve. Examination of the removed specimen revealed an unruptured gastric ulcer two centimetres in diameter on the lesser curve quite close to the pylorus. Histological examination of this ulcer proved it to be simple. The patient's subsequent convalescence was uneventful and he was discharged from hospital on December 13.

The patient has since been followed up, and when last examined on February 10, 1951, he was symptom-free. He had experienced no pain or digestive disturbance since he left hospital. He adhered to an ulcer diet for nine months after operation and had frequent small meals. Subsequently, on his own initiative, he abandoned the dietetic restrictions and found that he was able to eat anything at all. He was capable of performing manual labour and was maintaining his weight at the pre-operative level.

Comment.

This report is presented with the object of placing on record a case in which the perforation was so large that simple closure could not be successfully undertaken. Immediate partial gastrectomy appeared the best solution to the problem. Emergency gastrectomy for gastro-duodenal hæmorrhage has become a well-established procedure; but

emergency gastrectomy for perforated ulcer is still a comparatively rare procedure. In the case reported above, the perforation was so extensive that closure was impossible and there appeared to be no alternative to subtotal gastrectomy. As a general rule simple closure of the perforation is to be advocated, as rupture is a grave complication of peptic ulceration and the surgeon's primary consideration is to save his patient's life. However, in recent years both conservative treatment and radical resection have had their advocates. Hermon Taylor in 1946 reported a series of 28 cases of perforated ulcer treated conservatively with four deaths. His paper stimulated interest in the non-operative treatment of ruptured ulcer, and several small series have been reported since. In this connexion Professor Ian Aird (1949) writes as follows:

> Conservative treatment which comprises rest, suction drainage of the stomach and intravenous saline, is adopted only in centres where it has not been possible to reduce the operative mortality below 20 per cent. Conservative treatment cannot compete with the fatality rate of 5 or 6 per cent which is attained when diagnosis is early and operative treatment prompt and expeditious.

Tanner (1950) considers that the case for the conservative method has not been proven, and concludes: "Certainly, good risk cases are occasionally lost from persistent leakage when non-operative treatment is employed."

On the other hand, gastric resection has also had its advocates. The first radical operation appears to have been performed by Keetley (Cope *et alii*, 1938), who in 1902 successfully performed pylorectomy for a large perforation near the pylorus. By far the largest series yet published is that of Yudin (1939), who reported his own results in 937 cases of gastro-duodenal perforation treated by resection with a mortality rate of 8·9%.

In both the United Kingdom and the United States of America surgeons have been cautious in adopting emergency gastrectomy for ruptured ulcer, and it is likely that this procedure will be reserved for patients treated in clinics specializing in gastric surgery. In point of fact, in Avery Jones's (1953) own clinic, up to the middle of 1952 only 30 patients had been submitted to partial gastrectomy. There are obvious contraindications to radical surgery, such as senility, serious coincidental disease and general septic peritonitis. Yudin advised against gastrectomy if more than twelve hours had elapsed since perforation. On the other hand, certain circumstances may prevail under which emergency gastrectomy must be seriously considered. The first of these are (*a*) coincident perforation and hæmorrhage, and (*b*) suspicion that a gastric perforation is not due to a simple ulcer, but is carcinomatous. Doll (1950) has found that 8% of gastric perforations in men were in fact cancerous. Avery Jones recently stated that in his clinic there was an increasing tendency to perform emergency gastrectomy for ruptured gastric ulcer on this account. This applies particularly to men over the age of forty years. Two other circumstances in which emergency gastrectomy is likely to be needed are (*c*) the presence of massive perforations which cannot be closed satisfactorily (as in the case reported above), and (*d*) a history of more than one year's dyspepsia in a case of proven ulcer. (Avery Jones considers that this justifies emergency gastrectomy.) Most general surgeons, in dealing with such a case in which the perforation proved to be duodenal at operation, would be satisfied with simple closure and decide later whether elective gastrectomy was indicated according to the patient's subsequent progress.

The rather popular belief that perforation cures the ulcer has been exploded by Illingworth *et alii* (1946), who recorded the follow-up investigation of 733 patients in the Glasgow area. Within one year 40% had recurrence of ulcer symptoms (mild in half but severe in half), and within five years 70% had relapsed, symptoms being severe in 50%. It is therefore apparent that 30% of patients were symptom-free and 20% were in reasonable comfort on a medical régime. Hence it would be unjustifiable to submit all patients to emergency gastrectomy even when circumstances are propitious, as it would be unwarranted in half the cases. It has been established that there are regional differences in the behaviour of peptic ulcers, and it may be that in Australia the satisfactory long-term results are at an even higher level.

Summary.

1. A case of extensive perforation of a duodenal ulcer treated by subtotal gastrectomy is reported.

2. Modern trends in the treatment of ruptured gastric and duodenal ulcers are briefly discussed.

References.

AIRD, I. (1949), "Treatment of Perforation of Peptic Ulcer", in "Companion in Surgical Studies", 612.

COPE, V. Z., FLINT, E. R., and GALLOWAY, R. L. (1938), Discussion on treatment of perforated peptic ulcer, *Proc. Roy. Soc. Med.*, 31: 465.

DOLL, R. (1950), "Perforated Carcinoma of Stomach Simulating Perforated Gastric Ulcer", *Brit. M. J.*, 1: 215.

ILLINGWORTH, C. F. W., SCOTT, L. D. W., and JAMIESON, R. A. (1946), "Progress after Perforated Peptic Ulcer", 1: 787.

JONES, F. AVERY (1953), "The Management of Complications of Peptic Ulcer", M. J. AUSTRALIA, 1: 51.

TANNER, N. (1950), "Surgery of Peptic Ulcer", in "Techniques in British Surgery", edited by R. Maingot, 404.

YUDIN, S. J. (1939), *Int. Chir.*, 4: 219.

"DEADLY NIGHTSHADE" POISONING.

By W. D. GIBBONS,
Rockhampton, Queensland.

Clinical Record.

THE patient, a girl, aged three years, awakened at 6.10 p.m., grunted, frothed at the mouth, and lapsed into unconsciousness. The jaw was tightly clenched, and respiration was said to have ceased in a few moments. Artificial respiration of some sort was carried on by the father pending my arrival.

On examination of the child at 6.15 p.m. the respirations were rapid and shallow; the child was conscious and heavily flushed. The only other abnormal signs were as follows. Her temperature was 104·8° F., her skin was hot and perfectly dry. Her left tonsil was enlarged and there were some small left cervical glands. (The child had been treated for tonsillitis with cervical adenitis, and treatment had ceased one week previously.) The pupils were normal.

Inquiry revealed that the child had had some green berries in her purse that morning (found and discarded at about 10 a.m.). She admitted having chewed some of these. The berries were growing in an adjacent back yard, and were recognized as "deadly nightshade".

The provisional diagnosis of acute belladonna poisoning was made.

The stomach was not washed out, as at least eight hours had elapsed. A hypodermic injection of 0·25 milligramme of "Prostigmin" was given immediately (6.30 p.m.). Vomiting occurred thirty minutes after this injection, and mandarin, which had been ingested at 10 a.m., was returned.

On the following day an enema was given along with half a grain of calomel, and 0·25 milligramme of "Prostigmin" by hypodermic injection. The temperature remained in the vicinity of 103° to 105° F.

A second enema was given on the third day after her admission to hospital, when it was realized that absorption from the gut was still continuing. Very mild neck stiffness was noted at this stage.

The patient was discharged from hospital six days after her admission, having been febrile but otherwise fairly well for five days. She was perfectly well when reviewed a week later.

Comment.

I understand that the green berries of "deadly nightshade" contain large quantities of a group of alkaloids collectively referred to as "solanine". A mydriatic factor may or may not be present. The ripe berries are apparently

reasonably innocuous, as I recall eating these in my younger days. I would welcome any comment.

Reference.

WEBB, L. J. (1948), "Guide to the Medicinal and Poisonous Plants of Queensland", Bulletin Number 232, C.S.I.R.O.

Reviews.

Konversion und Reversion klinischer Neurosen: Ein Beitrag zur Pathologie der Furcht Versuch einer Neurosenkunde aus der Praxis des Internisten. By Hans Rehder, M.D.; 1953. Köln: Arzte (Publishers). 8¼" × 6", pp. 232. Price: 12.90 marks.

THE book is written on the experience of forty years. It is based on the doctrines of the German and Swiss psychiatrists Kretschmer, Hoche, Dubois and Bing. It is intended especially for practitioners and clinicians who have to treat patients in the early phase of their neuroses in everyday practice. The author believes that conversion, developing into a neurosis, can be caused to revert; his basic thesis is: "He who has been converted, can be reverted." The doctor has to acquire an exact knowledge of the situations and reactions which have led to neurosis. For the understanding of conversion and reversion the author gives a large number of short histories of patients with depression, neurasthenia, phobia, fright neurosis, hysteria and hypochondria. He is firmly convinced that in the analysis of the reactions which have induced the neurosis the doctor has to make the patient pass through them once more, and in sympathetic understanding the doctor has to pass through them too. He puts aside psychonanalysis, every kind of mental test, the explanation of dreams; in his opinion psychoanalysis cannot clarify the development of neuroses. The book is interesting, but not easily readable; on the contrary it is rather tiresome and not equal to up-to-date English and American books on psychiatry and psychotherapy.

A Guide to Human Parasitology: For Medical Practitioners. By D. B. Blacklock, C.M.G., M.D. (Edin.), D.P.H. (London), D.T.M. (Liver.), and T. Southwell, D.Sc., Ph.D., A.R.C.Sc., F.Z.S., F.R.S.E., revised by T. H. Davey, O.B.E., M.D. (Belf.), D.T.M. (Liver.); Fifth Edition; 1953. London: H. K. Lewis and Company, Limited. 10" × 6½", pp. 236, with 123 illustrations, three in colour. Price: 25s.

THERE have been five editions and seven reprintings of this book since its first publication in 1931. This clearly indicates its value and popularity. The success of the latest edition is assured, since it continues to be the only English text-book of its class in the field of human parasitology. It fills the demand by both students and workers in parasitology for a reference book which is easy to use quickly, and which contains adequate information for routine practical purposes.

While the general form of the book has been retained, the entire text has been completely revised, modified and brought up to date. Some chapters have been considerably expanded, others have been shortened and a few remain unaltered from the earlier edition. Many of the comparative tables, especially in the helminth section, have been omitted. This, in many cases, is regrettable, as many workers and particularly students find tabulated information very useful.

The new and revised plates are excellent. Plate II includes a greatly increased number of figures, of both trypanosome and malarial parasites. The figures are improved and resemble more closely the parasites as they are seen in stained preparations. Plate III, with its covering guide sheet, is a welcome addition which will be of inestimable value for teaching purposes. While Plate I would have been better placed opposite to page 54, Plates II and III may have been better arranged at the ends of their respective chapters.

Among the figures most worthy of note is the new Figure 21 on the microscopic constituents of fæces. By the retention of the series of diagrams illustrating the life-histories of the helminths on pages 202 to 217, much of the character of Blacklock and Southwell has been kept. Although these serve a function very well, similar diagrams arranged as a cycle could perhaps have been of greater value and appeal.

On the whole, the fifth edition of this book has been thoroughly and soundly revised. It will be welcomed to the shelves of all parasitologists. Teaching personnel particularly will be grateful to those who, through many hours of hard and tedious work, have made the publication of this new edition possible.

Pediatrics. By L. Emmett Holt, junior, and Rustin McIntosh; Twelfth Edition; 1953. New York: Appleton-Century-Crofts, Incorporated. 10" × 7", pp. 1502, with 271 illustrations. Price: $15.00.

THIS volume represents the twelfth edition of the well-known "Diseases of Infancy and Childhood", originally written in 1896 by the late L. Emmett Holt, Professor of Diseases of Children at Columbia University. The field of pædiatrics has long since grown beyond the scope of single authority, and multiple authorship is a virtual necessity. Holt and McIntosh share their task with 72 collaborators. Amongst many famous names, Hattie Alexander writes on influenzal infections, Dorothy Andersen on the pancreas, Helen Taussig on diarrhœa, Thorn on the adrenals, and Ford on the nervous system. However, as the authors remark in their preface, they have attempted to avoid the chief difficulties of multiple authorship by synthesizing their own viewpoints with that of their collaborators—"a unity of concept which has been attained at times after much profitable discussion".

The first chapter is devoted to the care of the normal infant and general aspects of growth and development, and there is an excellent 50 page section entitled "Peculiarities of Disease in Children". One notices that the artificial feeding of infants has been simplified in the past quarter-century, and that "in the United States evaporated milk is now more widely used for infant feeding than any other product, combining as it does the virtues of maximum digestibility, sterility, uniformity of composition, convenience of preparation and cheapness".

The section on "Psychopathologic Problems" is good, but rather too short (35 pages), whereas the chapter on "Nutrition and Nutritional Disturbances" (117 pages) could with advantage have been curtailed. There is a new chapter on "Collagen Diseases", which includes rheumatic fever, and an excellent account of pink disease, for which the authors accept sensitivity to mercury as the ætiological factor. Another useful section is devoted to such affections of the eyes as commonly present themselves to the pædiatrician.

In the management of *diabetes mellitus* the authors favour the plan, generally adopted in Scandivanian countries and becoming popular in Australia, of a free diet with the insulin dosage adjusted to avoid ketonuria while permitting almost continuous glycosuria.

This is a splendid book, clearly written and containing an enormous amount of material. It is up to date and authoritative, and ideal for the child specialist and for every medical library.

Basic Pathology and Morbid Histology. By D. B. Cater, M.A., M.D. (Cantab.), F.R.C.S. (Eng.); 1953. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 9½" × 6¼", pp. 338, with 266 illustrations. Price: 42s.

As the author points out in the preface, this book is written for students and not for professors. It is perhaps unfair, therefore, to ask for an impartial review from anyone but a student. However, an attempt has been made to sum this book up, the readers to whom it is addressed being kept in mind.

The book is divided into three parts, dealing respectively with the reaction of the body to infection, diseases due to breakdown of physiological mechanisms, and tumours.

Throughout the first part free use is made of analogy to warfare, which is very apt in places and should help the student to grasp the underlying principles of inflammation. This part is written in a racy, readable style and keeps strictly to the terms of reference—namely, basic pathology.

In Parts II and III, not only the style but also the choice of material differs. Some of the essential facts of basic pathology are omitted, whilst subjects of a non-basic character are included. Why, for instance, is there not a section on pigmentation when many forms of congenital heart abnormality are introduced and meningioma is subdivided into five different types? In the last two chapters, the cause of neoplasia is discussed in a most unsatisfactory manner, the student being presented with a mass of unnecessary detail instead of being led by the hand—as is done in Part I—and having pointed out along the way the more important items of interest.

There are 264 figures made up of *camera lucida* histological drawings, most of which are in colour, pencil sketches of macroscopic specimens, and diagrams of varying complexity. With only a few exceptions, the *camera lucida* drawings are clear and would be most helpful to the student in his early stages. Many of the pencil sketches, on the other hand, serve no useful purpose and could with great advantage be replaced with photographs.

To sum up, this book will have a limited appeal to students; but it should be read only in conjunction with, not to the exclusion of, other already established and proven books covering the same ground.

Clinical Psychiatry: For Practitioners and Students. By Ian Skottowe, M.D. (Glasgow), M.R.C.P. (London), D.P.M. (London); 1953. London: Eyre and Spottiswoode. 8¾" x 5½", pp. 407. Price: £1 16s.

THIS book is offered as a psychiatric contribution to the "Practitioner" series of publications. The essentials of such a work would seem to be simple, direct, and explicit thoughts with a minimum of technical phraseology and specialist jargon, after the manner in which the physician addresses his remarks and explanations to the patient and his relatives. In this respect, then, this book is easy to read and the continuity of thought is easy to follow.

Some time is spent in developing the concept of the unity of human illness, which in turn is considered as a fault of adaptation. The book covers the more recent concepts of psychological medicine.

The handling is chiefly along practical descriptive lines, with a minimum of space given to the more theoretical dynamic psychology.

A useful chapter is directed to a study of the principles of treatment. This would be very helpful in orientating the student in the value of the several psychiatric treatments available for a particular disease.

The physician will have no difficulty in identifying many of his most distressing and tedious patients.

Like all good text-books, this book contains all the worthwhile information available. The author is to be congratulated on producing such a useful work.

Statistical Tables: For Biological, Agricultural and Medical Research. By Ronald A. Fisher, Sc.D., F.R.S., and Frank Yates, Sc.D., F.R.S.; Fourth Edition; 1953. Edinburgh: Oliver and Boyd. London: Macmillan and Company, Limited. 11½" x 9", pp. 138. Price: 21s.

THE new edition of the well-known and popular "Statistical Tables" of Sir Ronald Fisher and Dr. F. Yates continues the policy of the previous editions. Tables are given for all the commonly used statistical tests and for many of the simpler mathematical functions; but the authors have avoided supplying tables which would be of value only in very restricted fields. The result is a most useful book of tables for all those engaged in the statistical analysis of data in agricultural, medical and many other fields.

New material in this edition consists of some tables useful in the analysis of time series, in genetics and in the design of experiment. The format, binding and printing are excellent.

Handwriting Analysis: As a Psychodiagnostic Tool: A Study in General and Clinical Graphology. By Ulrich Sonnemann, Ph.D., with a foreword by Bela Mittelmann, M.D.; 1952. London: George Allen and Unwin, Limited. 9" x 5½", pp. 286, with 247 illustrations. Price: 35s.

SINCE Ludwig Klages and Robert Saudek published their investigations into the psychology and character analysis of handwriting a quarter of a century ago, there has grown an extensive literature to which this book is the latest addition. Like Omar Khayyám, the graphologist attempts to define "up-and-down", with a view to interpreting the psychological characteristics of the writer. Rhythm, regularity, intergration, fluctuation, velocity, impact, pressure, binding, slant and amplitude are only a few of the components of script which are studied by the graphologist.

Downey's will-temperament test, based on a simpler analysis of handwriting, appears to have found little favour amongst clinical psychologists, and it is doubtful whether the much more complicated system described by Dr. Sonnemann will justify the necessary expenditure of time. The text is copiously illustrated by specimens of handwriting including the signatures of Hitler and Mussolini. There is no reference to "doodling", which at least offers a clue to the phantasy life. Psychiatrists are, of course, well acquainted with the distinctive features of the handwriting of manics, melancholics and schizophrenics, and of patients with cerebral degeneration. Dr. Sonnemann offers suggestions also regarding the diagnosis of psychoneurotic and inadequate personalities. His book may be accepted as an authoritative guide to the psychological interpretation of handwriting.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Clinical Chemical Pathology", by C. H. Gray, D.Sc., M.D., M.R.C.P., M.R.C.S., F.R.I.C.; 1953. London: Edward Arnold and Company. 7½" x 5", pp. 144, with 17 text figures. Price: 10s. 6d.

This is based on lectures to medical students, but the author hopes that it will be of value also to resident medical officers and registrars.

"Health Services for the Child", by Edward R. Schlesinger, M.D., M.P.H., with a foreword by Herman E. Hilleboe, M.D.; 1953. New York: McGraw-Hill Book Company, Incorporated. 9½" x 6½", pp. 422, with 12 text figures. Price: $7.50.

This book is written for the practising doctor; its purpose is to give an integrated picture of health services for mothers and children.

"The Megaloblastic Anæmias", by L. J. Davis, M.D., F.R.C.P., F.R.C.P.E., F.R.F.P.S.G., F.R.S.E., and Alexander Brown, M.D., F.R.C.P.E., F.R.F.P.S.G.; 1953. Oxford: Blackwell Scientific Publications. 8½" x 5½", pp. 124, with 12 illustrations. Price: 21s.

A monograph addressed to the general physician rather than the hæmatologist.

"A Textbook for Midwives", by Margaret F. Myles, S.R.N., S.C.M., H.V.Cert., Sister Tutor Cert., M.T.D., with a foreword by Jean P. Ferlie, O.B.E., R.G.N., S.C.M., 1953. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 6", pp. 688, with 380 illustrations. Price: 42s.

The aim is to present obstetrics from the midwife's point of view and in a form suitable for the pupil midwife.

"Social Medicine", by S. Leff, M.D., D.P.H. ("Survey of Human Biology", edited by S. A. Barnett: Number One); 1953. London: Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 9" x 6", pp. 310, with about 20 graphs. Price: 28s.

An attempt is made to give a general survey which will guide explorers in the field of social medicine.

"The Psycho-Analysis of Artistic Vision and Hearing: An Introduction to a Theory of Unconscious Perception", by Anton Ehrenzweig; 1953. London: Routledge and Kegan Paul, Limited. Sydney: Walter Standish and Sons. 8½" x 6", pp. 304, with 10 plates and 25 text figures. Price: 25s.

"The book deals with the inarticulate form elements hidden in the unconscious structure of a work of art or—what comes to the same thing—with the unconscious structure of the perception processes by which we actively create or passively enjoy the unconscious form elements."

"Textbook of Gynecology", by John I. Brewer, B.S., M.D., Ph.D.; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6½", pp. 550, with 146 illustrations. Price: £5 7s. 6d.

An attempt has been made to present the subject from the clinical rather than from the didactic point of view.

"Bailey's Text-Book of Histology", revised by Philip E. Smith, Ph.D., Sc.D., and Wilfred M. Copenhaver, Ph.D., with the assistance of Dorothy D. Johnson, Ph.D.; Thirteenth Edition; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 6½", pp. 794, with 442 illustrations, a few in colour. Price: £4 16s. 9d.

The first edition was published in 1904.

The Medical Journal of Australia

SATURDAY, FEBRUARY 13, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

HEART DISEASE IN INDUSTRY.

THE subject of heart disease in industry, which was discussed at a meeting of the New South Wales Branch of the British Medical Association last August, is one of unusual difficulty, and therefore of the greatest interest. The papers and discussion, published in this issue, should be read with care by every reader of this journal. Dr. F. A. E. Lawes gave a comprehensive review of the subject and also included some of his own opinions. Dr. U. L. Brown spoke from a wealth of personal experience and he shows himself as an industrial medical officer imbued with both wisdom and common sense. When we think of heart disease in industry, we naturally turn our minds to the question of compensation for injury. It will be wise, however, to divert our attention from this aspect of the subject and to consider the academic question whether physical effort can inflict damage on a heart. For this reason, it is perhaps a pity that no pathologist experienced in the subject was asked to present a paper at the meeting. This omission was to a large extent made good by the many references to the literature by the various speakers. The bibliography at the end of Dr. Lawes's paper will indicate where readers may, if they wish, obtain further ideas on the subject.

Most people will agree that ordinary industrial effort will not produce damage in a normal, healthy heart. Most authorities agree that the heart which receives damage has been the subject of a pathological process affecting the arteries of the heart wall and thus its musculature. Recovery often follows what we may call coronary occasions, and it is by no means uncommon during the conduct of post-mortem examinations after a fatal coronary episode, to find not only disease of the coronary vessels but a healed lesion from a previous episode. Dr. Lawes stated that he did not believe that coronary occlusions occurred as a result of effort. At the same time, he said that one could conceive of the occurrence of infarction of muscle or even death of muscle from prolonged anoxia. Here we may turn to what Dr. Brown said, that *angina pectoris* had for its basis a relative cardiac ischæmia—an inadequate blood supply to the myocardium relative to the demand made upon it. He added that if the blood supply to an area of myocardium then failed altogether and no alternative blood supply was available, that area of the myocardium must die, and myocardial infarction would occur—either the blocked vessel was occluded by atheroma *per se* or the blocking was completed by a thrombus. Here it will be convenient to refer to the well known work of A. M. Masters, S. Dack and H. L. Jaffe which was reported several years ago. Three long discussions were devoted to this work in these columns as long ago as December, 1940, and January, 1941. It may be recalled, perhaps, that Masters, Dack and Jaffe discussed no less than 1620 attacks of coronary occlusion, It should be remembered, however, that they insisted on distinction between angina and coronary inefficiency with infarction on the one hand and coronary occlusion on the other. They dealt only with cases of the latter group. In their investigations, Masters and his co-workers inquired into the activities of the patient, not only at the time of the attack, but during the preceding twenty-four hours and also for the four weeks prior to the occurrence of the attack. It is interesting to note that 50 patients who were seeking compensation were excluded since it was found that their histories were unreliable—the history given by the patient in the hospital after the attack and that given at the compensation hearing months later revealed that the former story was often entirely at variance with the latter. Some observers have maintained that these attacks often occur while the patient is in bed or at rest. Masters and his associates found that 52·2% of the attacks investigated by them occurred while the patient was asleep or at rest; 21% occurred during routine activities; 15% occurred while the patient was walking at an ordinary pace; and 9% occurred during moderate activity. Only 2% were associated with unusual activity. Masters, Dack and Jaffe pointed out that these figures corresponded quite closely to the portion of the day spent by the majority of people in each activity mentioned. They stated that it might reasonably be concluded that the onset of the occlusion was not influenced by the activity or lack of activity of the patient at the time. They held that the association of occlusion with the activities of the patients just mentioned was merely incidental. They went so far as to insist that the association of unusual activity with the attack in only 2% of cases excluded effort as a factor. No less than 70 of their patients, prior to the occurrence of occlusion, had been bedridden on account of chronic illness. Of the patients in their series, 36% were manual workers and labourers, 9·5% were "white collar and office workers", 8·5% were professional persons, 11% were business men, 20% were housewives, 9·5% were "retired". These percentages, they pointed out, corresponded to the occupational distribution in New York City. Though it is neither possible nor desirable to traverse the whole ground

of the work described by Masters, Dack and Jaffe, two points in their arguments should be mentioned. They pointed out that it was well known that increase in arterial pressure was not transmitted to the peripheral capillaries, and there was no proof that an increase in aortic pressure was transmitted to the capillaries arising from the lumen or adventitia of the coronary artery. They also referred to the failure of Winternitz and his co-workers to produce rupture of intimal capillaries when they injected the coronary vessels of human hearts at a pressure of 500 to 1000 millimetres of mercury, pressures far beyond any attained during life.

If we bear in mind the observations reported so long ago by Masters, Dack and Jaffe, it is worth while to recall observations reported in *The Lancet* of November 21 and 28, 1953. These were described in these columns on January 2, 1954. The work was carried out by J. M. Morris, J. A. Heady, P. A. B. Raffle, C. G. Roberts and J. W. Parks. These observers made an investigation into the cardiac crises of omnibus conductors and drivers in London. They found that the conductors, whose jobs obviously involved considerable physical activity, had a different experience of coronary diseases from the drivers, whose jobs involved less physical activity. They made an analysis of the experience of postal workers and civil servants, and found a similar trend when postmen were compared with sedentary workers. The "provisional hypothesis" stated by them was as follows:

> Men in physically active jobs have a lower incidence of coronary disease in middle age than men in physically inactive jobs. More important, the disease is not so severe in physically active workers, tending to present first in them as *angina pectoris* and other relatively benign forms, and to have smaller early case fatality and a lower early mortality rate.

It is to be noted that among the workers included by Morris *et alii* are those deliberately excluded from the observations of Masters and his colleagues. This should not really affect the point at issue. As mentioned in our issue of January 2, 1954, Morris *et alii* raise a number of questions in their paper, and none is answered with finality. To begin with, the possible association and perhaps confounding of psychological with physical factors has to be considered. Another point is the possible effect of physical activity on the coronary circulation. One important observation made was that men engaged in more active work appeared to have not merely a lesser total incidence of coronary disease, but also within this feature more angina. By and large it would appear that physical activity is advantageous to workers from the cardiac point of view. In these circumstances, the appearance of anginal pains would be an indication for investigation and the institution of precautionary measures. Factors other than physical effort and mental activity apparently are brought into play, and a good deal of research is yet needed before the chart may be described as complete. The basis of the whole subject is obviously the state of the coronary arteries, and if coronary occlusion and other coronary disasters are to be prevented, we must learn how to preserve the arterial system, particularly of the heart, in a healthy condition. It will probably be said that with the stress of modern life this would be impossible; but research may yield secrets of which we have at present no inkling, and we must learn to travel hopefully.

Current Comment.

ADRENALECTOMY IN THE TREATMENT OF ADVANCED CARCINOMA OF THE BREAST.

THE case reported in this issue of the journal by Dr. Kathleen Cuningham is one of most absorbing interest. Dr. Cuningham gives an account of her treatment by adrenalectomy of a patient who was suffering from extensive metastases from carcinoma of the breast. The result achieved is most remarkable. Dr. Cuningham does well to point out that the future of this patient cannot be foretold; in other words, that what has been produced is probably a remission of the disease. In the course of her report, Dr. Cuningham refers to an article which has been published by C. Huggins and T. L-Y. Dao. This contribution, which appeared in *The Journal of the American Medical Association* of April 18, 1953, will help to put Dr. Cuningham's case in its proper perspective.

Huggins and Dao report that during 21 months of 1951 and 1952, 55 patients with mammary cancer were treated by adrenalectomy, two men and 53 women. In this series, there were three deaths within thirty days of the operation (all the patients who died were women); 50 women remained, therefore, for discussion on the value of the treatment adopted. In a quarter of the cases adrenalectomy was combined with oophorectomy. In 19 cases the adrenals and ovaries were removed in two stages, the operations being about two weeks apart. Huggins and Dao found that in the six cases in which the combined operation was performed, no added morbidity occurred and there were no fatalities. The authors point out that the women whose mammary cancers were treated by removal of the adrenals alone had no significant ovarian function. The age range was forty-four to seventy years, the average being fifty-two years. All patients who were fifty-two years of age or younger had had X-ray sterilization or oophorectomy at an earlier date. Before adrenalectomy each of the women had been subjected to various forms of endocrine treatment. At the time when Huggins and Dao wrote their article, nine of the patients had died of cancer one and a half to seven months after operation, and three patients had advancing disease. They think that in ten instances the patient had a regression of the disease of some magnitude. In another table are set out the results obtained in the treatment of 25 women after combined adrenalectomy and oophorectomy. The age range in this group was twenty-nine to fifty-nine years, the medium age being forty-three years. The three oldest patients were aged fifty, fifty-eight and fifty-nine years. Before adrenalectomy ten of the patients had had ovarian irradiation, and fifteen had been treated with testosterone. Despite these treatments, the disease was advancing in every instance. There were no immediate post-operative deaths. Eight patients subsequently died of malignant disease forty-seven days to sixteen months after operation; three patients, at the time of the reporting, presented evidence of advancing disease, and in four instances insufficient time had elapsed to allow evaluation to be made. Ten patients had had a remission of disease with objective evidence of improvement; the improvement occurred in spite of the previous failure of ovarian irradiation and the use of X rays with testosterone. The commonest areas for regression to occur were in the osseous and pleural metastases. In five instances in which a remission did not ensue after adrenalectomy and oophorectomy, the patient was treated subsequently with testosterone propionate, but no benefit was observed. Under the heading of duration of improvement, it is pointed out that 14 patients had been observed for from one to two years, either after adrenalectomy or after combined excision of ovaries and of the adrenal glands. Six of the patients were dead, and one patient was alive, but her condition was unimproved. Seven patients were still in remission; two of these had had adrenalectomy alone, while five had undergone the combined procedure. These women appeared to be in good health and were all somewhat obese, having gained from 10 to 20 kilograms in weight.

In regard to the two men who were treated for mammary cancer, it is stated that both had previously been subjected to orchidectomy. One patient, who had pulmonary and intracranial metastases, had a considerable decrease in the size of the pulmonary metastases after adrenalectomy, but the cerebral lesions advanced and he died seven months after the operation. The other man, who had extensive pleural metastases, had a profound regression of the lesion and was in good health eleven months after removal of the adrenal glands. Hæmorrhagic pleural fluid, which had been accumulating rapidly in this patient in spite of repeated thoracentesis before adrenalectomy, had disappeared and had not reformed since the operation. Cancer cells had been found in this pleural effusion.

Huggins and Dao comment on their cases and discuss probabilities of the *modus operandi* of the treatment. They state that the mechanism of the improvement after adrenalectomy in cancer of the female breast seems to be the withdrawal of critical amounts of hormones similar to those formed in the ovary. After adrenalectomy in women who have undergone oophorectomy, the excretion of œstrogen in the urine is abolished. Increased pituitary activity of the type that occurs in post-menopausal women is stated not to be incompatible with regression of mammary cancer. To discuss this matter further would lead to too much conjecture, and in the circumstances this does not appear to be justified. In the treatment of advanced cancer of the breast, what has to be thought of is the comfort of the patient. It does appear that adrenalectomy with oophorectomy is likely to prolong the lives of certain women with advanced mammary cancer. Increased experience will probably enable surgeons to determine which patients should be subjected to this treatment and which should have nothing but palliative forms of therapy. It must, of course, be pointed out that adrenalectomy is not a simple procedure, and that it calls for skill, not only on the part of the surgeon, but, as Dr. Cuningham's anæsthetic chart shows, on the part of the anæsthetist.

THE USE OF SALICYLATES IN GOUT.

A VERY interesting and important study in gout has been reported by F. G. W. Marson, from the Department of Therapeutics of the University of Birmingham and the Birmingham General Hospital, England.[1] This communication makes particular reference to the value of sodium salicylate in the treatment of gout. The suggestion that sodium salicylate should be used for this purpose is by no means new. In Allbutt and Rolleston's "System of Medicine", published in 1907, we read under the section devoted to gout that salicylates have been largely tried in the treatment of gout, both in Great Britain and on the Continent. The clinical evidence as to their efficiency is stated to be very contradictory. In the "Tenth Rheumatism Review", published in the *Annals of Medicine* of September, 1953, a section is devoted to gout and under the heading of "Treatment" we read the single sentence: "Intermittent administration of salicylate was advised." Marson's conclusions are so definite that medical practitioners should be made aware of them. Marson's study deals with acute and chronic gout, and covers some 115 patients. Of these, 32 suffered from chronic gout—28 males and four females. It is to these that we direct particular attention. Marson states that since urate deposition appears to be the all-important feature of chronic gout, treatment should be directed towards its prevention, and aim at maintaining a persistently lowered urate content of the body fluids to avert the precipitation of urate crystals. He goes on to observe that, theoretically, treatment may be directed either to reduction of the rate of uric acid formation in the body or to the administration of drugs which reduce the reabsorption of urate through the renal tubules. Many attempts at such therapy have been made, but with little success. Marson quotes Talbott, who wrote in 1949 that he believed that all that would be needed would be means to produce a permanent reduction of concentration of urate in body fluids. If the urate concentration could be maintained permanently in the normal range, gouty arthritis would never develop. Marson explains that uric acid is the end product of purine metabolism in man, and is derived normally from exogenous and endogenous sources in almost equal proportion. Isotope studies have shown that glycine and ammonia contribute nitrogen to the formation of uric acid, and that carbon atoms are derived from formate, glycine and carbon dioxide. From this it appears that in addition to purine, the dietary carbohydrates, fats and proteins are all potential precursors of uric acid. Marson states that in spite of the widespread belief that diet is an important causal factor in gout there has been little controlled investigation into the effects of dietary changes upon serum uric acid levels. It would be difficult to recall a text-book in which the treatment of gout was discussed and in which control of the diet was not mentioned as one of the *desiderata*. Here perhaps we may anticipate one of Marson's conclusions in his summary, that there is probably no justification for dietetic restriction. He comes to this conclusion because of experiments with diets of varying purine content, which confirm his belief that the serum uric acid content cannot be effectively controlled by the limitation of purine intake. This, however, is perhaps a digression from the question of sodium salicylate therapy, although it is of importance.

Marson states that it is known that certain drugs have a depressing effect on urate reabsorption. "After their initial administration in suitable dosage the urine uric acid is increased and the plasma uric acid decreased, there being no increase in glomerular filtration." The drugs which have this effect include salicylates, cinchophen, caronamide and "Benemid" given orally, and "Salyrgan", "Diodrast", glucose and phenol red given intravenously. Marson deals only with the prolonged use of sodium salicylates. He goes into the history of sodium salicylate administration and points out that Byasson in 1877 first showed that sodium salicylate produced an increased urinary excretion of uric acid. This observation was confirmed by other observers, one of whom pointed out that the salicylate had to be given in large dosage. In 1920 Graham recommended that sodium salicylate should be given to gouty people for two or three days each week or fortnight. He stated that it was useless to give this drug for longer periods, since it caused an increased output of uric acid for two or three days only. Jennings, in 1937, advised that sodium salicylate should be used in the prophylactic treatment of gout, and he treated his own patients with 80-grain doses every day for three or four days a week. He stated that by this means normal levels of uric acid in the blood were maintained. Bauer and Klemperer were unable to confirm Jennings's findings. One of the most recent workers on the subject is Gutman, who reported in 1950 that a dosage of salicylate sufficient to reduce the serum uric acid content could not be maintained for more than a few consecutive days because severe salicylism developed. Marson gave sodium salicylate only to patients suffering from chronic gout. It was dispensed in a fluid mixture containing an equal quantity of sodium bicarbonate, a flavouring agent and sodium sulphite as a preservative. Each dose was made up to half an ounce and the mixture was usually given three times a day; the total daily dosage varied from 60 to 140 grains. The aim was to regulate the salicylate dosage so as to maintain normal serum uric acid levels and to avoid serious toxic symptoms. Patients were admitted to hospital for the institution of continuous salicylate therapy. After preliminary investigation, including the performance of renal function tests, the salicylate was prescribed in an initial dosage of 30 grains three times a day. Symptoms of salicylism commonly occurred, but unless they were severe, patients were encouraged to endure them for a few weeks and the symptoms usually disappeared. While the patients were in hospital the serum and urinary acid levels were estimated every day. After the patients had been sent home they maintained continuous salicylate therapy and

[1] *Quart. J. Med.*, July, 1953.

attended a gout clinic at intervals of one to four weeks. At each attendance progress was recorded and the serum uric acid content was estimated. Considerable improvement in the patient's condition occurred in all cases during the period in which sodium salicylate maintained the serum uric acid at normal or nearly normal levels. Pain and stiffness of the joints disappeared from 20 of 28 patients. Several who had previously been crippled were restored to normal activity for the first time in many years. Tophi were noticed to disappear, ulcers to heal, joint destruction to be arrested, and the range of movements to be increased. The most striking proof that urate deposits were absorbed under this treatment was provided by radiological studies undertaken in four cases.

The question will naturally arise as to how long this strict regimen should be imposed. We read that cessation of salicylate therapy in a few cases has not resulted in an immediate return of symptoms, but that the serum urate level rises rapidly. The extent of such symptomatic remissions is as yet not known, and Marson thinks that it may be possible to maintain adequate control of the disease by the administration of salicylates for a long period of each year. He is undertaking further studies to elucidate this point. He emphasizes the fact that the continuous treatment by salicylates will not entirely prevent the occurrence of attacks of acute gout, but there is little doubt that such attacks are reduced in frequency and severity. He states that the acute aspect of the disease presents little difficulty, as the colchicine treatment of acute attacks is highly satisfactory. The final statement in Marson's discussion is that his present impression is that continuous sodium salicylate therapy constitutes the most practicable method of controlling the levels of serum uric acid in cases of chronic gout. It is to be hoped that this work of Marson's will be repeated by others and that the results will be reported.

LOBOTOMY.

IN 1935 Moniz introduced the procedure of lobotomy for the treatment of patients suffering from mental disorder. Since then it has had extensive trial in most parts of the world and has gained wide acceptance, although it is, as William Sargant[1] puts it, as yet obtaining only a hazardous social and medical respectability. According to Lawrence C. Kolb,[2] a survey made by the National Institute of Mental Health in the United States indicated that by the end of 1949 about 10,000 persons in that country had undergone lobotomy or some form of surgical ablation of the frontal lobes of the brain for mental illness. The number of operations of this type had increased annually during each of the five preceding years. The operation most used had been that of prefrontal lobotomy—in general, according to the procedure used by Freeman and Watts—but many modifications had been introduced which had changed the picture considerably and blunted the criticism made of the earlier operations. We shall refer to some of these presently. Kolb's paper, however, is concerned essentially with prefrontal lobotomy, of which he has had extensive experience over a period of fifteen years. He does not consider that the modified techniques have been used over a sufficient period to allow a satisfactory evaluation of the therapeutic potentialities, and so refrains from discussing them. So far as prefrontal lobotomy is concerned, he considers that in the past five years data have accumulated that allow a much better appraisal of its value. Many of the questions answered in 1947 have now been clarified. His broad judgement is that prefrontal lobotomy has proved to be a useful therapeutic procedure in the treatment of certain emotional disorders and in cases of intractable pain. It is symptomatic therapy that has proved most effective in relieving destructive drives, chronic depressions and obsessions, hypochondriasis, impulsiveness and overactivity. It seems to promise return to the community of a greater number of patients with chronic mental disease who have been in hospital for more than two years than does any other therapeutic procedure. On the other hand, relapse, with full recurrence of the symptoms of the original mental illness, may occur after prefrontal lobotomy, and there is evidence that patients who have undergone lobotomy are more sensitive to environmental stress after operation than before. Moreover, most patients show evidence of deficit due to the lesion of the frontal lobe, but this defect is described as being often not so disturbing as the emotional illness they previously had. Other complications frequently encountered are convulsions and urinary incontinence and urgency.

This and many other points in Kolb's paper are honest blunt criticism of a procedure that is not lacking in opponents, but the experience gained through the operation has been very considerable, and in any final assessment there will probably be a good deal on the credit side. Just the same, it is interesting to learn from Walter Freeman,[1] who with J. W. Watts was largely responsible for what has been the standard operation of prefrontal lobotomy, that he has abandoned the use of this procedure almost entirely in the past three years. The reason for its abandonment is stated to have been the high incidence of fatalities and complications. It has been supplanted by transorbital lobotomy. Freeman discusses the results, and in particular the hazards during operation and undesirable sequelæ from his experience in 702 operations for prefrontal lobotomy and 1303 operations for transorbital lobotomy. He states that while there is slightly higher risk of fatal hæmorrhage with transorbital lobotomy, the incidence of other causes of operative fatality is so low that the mortality is only half that for prefrontal lobotomy. Long-term follow-up in the series of patients treated by prefrontal lobotomy by Watts and himself shows a wide distribution of causes of death not linked with operation; there is a preponderance of deaths from heart disease. On the other hand, in the transorbital lobotomy series a larger number of late deaths are attributable to malignant disease, a circumstance readily explained by the fact that transorbital lobotomy is an effective palliative measure for the pain and suffering of the patient in a terminal cancer state, while prefrontal lobotomy is too drastic a measure for this purpose. Physical complications, such as hemiparesis, seizures and incontinence, are ten times as prevalent after the major operation, that is to say, prefrontal lobotomy, as after the minor transorbital operation. Socially undesirable traits also are ten times as common after prefrontal lobotomy as after transorbital lobotomy. It should be noted that the purpose of the paper is to evaluate the different types of operations from the point of view of safety and not from that of effectiveness, but Freeman in his concluding remarks states that the hazard of performing lobotomy for the relief of mental disorders is traced in its proper perspective by noting the hazard of not performing the operation, the hazard of delay. To illustrate this, he has prepared a chart, based upon a study of more than 1000 patients who have been followed beyond the time of stabilization for periods ranging up to more than five years after transorbital lobotomy and more than fifteen years after prefrontal lobotomy. In this chart the good results are plotted against the total duration of stay in hospital before lobotomy was performed. By "good result" is meant that the patient not only is out of hospital, but is actively engaged in some type of useful activity, earning a living, keeping house or going to school. The chart shows that there are two chances out of three of returning to useful activity for patients treated by lobotomy who have had less than six months in hospital. Chances are about even for those with one year in hospital; but from then on the chances decrease considerably, so that of patients with chronic illness only one in ten can expect to return to some useful work outside the hospital.

Two recent English reports are more particularly concerned with the modified operations. Since 1942 William Sargant (whose paper was referred to earlier) has had the opportunity of observing patients who have undergone

[1] *Brit. M. J.*, October 10, 1953.
[2] *J.A.M.A.*, July 18, 1953.

[1] *J.A.M.A.*, June 6, 1953.

many of these modified operations, more particularly for neuroses. He states that now the use of these "limited cuts" has entirely transformed clinical possibilities of leucotomy. By 1944, with Slater, he was already able to stress as a result of their early finding that "for each patient a minimum of brain tissue must be destroyed that will produce the desired clinical effect" and that "the operative skill of the surgeon is required to ensure that the operation is not so extensive and destructive" as to produce unnecessary and undesirable sequelæ. The various modified procedures that have been attempted are many, and it is not necessary to go into the details of individual procedures here. However, Sargant states that it is no longer as easy to believe those who have suggested that the thing that really matters is the quantitative amount of brain damage done in the prefrontal lobe rather than its actual site. His own clinical impressions increasingly support Fulton's work on animals that the more the surgeon keeps to the inferior medial quadrant, the safer things are. The further the surgeon ventures from this area, the more likely is he to run into undesirable sequelæ, though the farther forward the damage, the better. Especially for patients with broad foreheads, the cutting of fibres in this area by a lateral approach may be difficult, unless special techniques are employed, and many patients still showing tension after an apparently full cut may be, in fact, those in whom important fibres in this quadrant have unfortunately been missed. Quantitative factors in the cut are also very important, but Sargant believes that it will be finally shown that to obtain the best clinical results with a minimum of undesirable sequelæ in neuroses, the site of the operation is more important still. That may be the reason, he states, why Freeman's new deep frontal cut, in which the transorbital route is used, is apparently achieving many successes in the United States, since it produces its maximum damage in just this lower medial area of the frontal lobe. There are also far fewer psychotic patients than is generally supposed who need to pay the price of a full posterior cut for their release from a mental hospital, though the deliberate production of emotional blunting is necessary in some. According to Sargant, one can now promise the properly selected patient that, barring accidents, he will certainly not come to great harm, though total relief of distressing symptoms may not be as dramatic and certain. With increasing surgical skill and modified operations accidents are becoming relatively fewer; and, like death from the operation, because of their increasing rarity, they need not now weigh too prominently in the final decision. Provided the right clinical indications are present, operations can be performed if necessary on patients with intractable psychosomatic states, anxiety hysteria, and anxiety and depressive states associated with obsessive personalities. Such patients will also often do much better than severe obsessionals for whom a more extensive operation may be necessary, as for the chronic psychotic, to remove symptoms making life intolerable. One finding is described as having been consistently true in the past ten years: "The more worried you become about subjecting a well-preserved neurotic patient to a modified leucotomy because the basic personality remains good, the better he will do." The unhappy results will be in patients for whom an operation has been recommended more lightheartedly because their personality was not so well preserved, and there was so much less to lose. Patients should be chosen from the best rather than the worst clinical material.

Sargant also lists a number of other principles that need to be observed if disaster is to be avoided. He stresses the importance of having all other methods of treatment available for trial first, and the value of a long period of preliminary study of the patient as an in-patient. One important point should be noted in the light of some of the severe criticisms of the results of earlier operations. Sargant states that if anterior and limited cuts are used, conventional, religious and ethical values are rarely irrevocably lost. Nevertheless, he points out that the previous personality must be taken into consideration in final judgements on such issues, as socially undesirable traits not recognized before operation can be enhanced in certain instances even by limited cuts.

One particular modified operation, orbital leucotomy, comes in for interesting discussion by P. Macdonald Tow and Walpole Lewin from the Nuffield Department of Surgery in the University of Oxford.[1] Orbital leucotomy is an open operation and consists of a horizontal incision in the white matter just above the orbital cortex. Tow and Lewin state that of 20 patients with various psychiatric disorders operated on two years ago, seven have recovered from the disorder, and eight more have improved. There was no death due to the operation. They discuss the issue raised by Sargant, namely the relative importance of the amount of cerebral tissue destroyed and the particular area involved. They state that there is still no sure clinical guide to what exactly should be cut in individual clinical syndromes, and there is still no decisive evidence about the relative merits and demerits of the different incisions. In practice there has developed an attitude of caution towards the major operation, but with great uncertainty about what to use in its place. They refer to a suggestion of Sargant and Slater that where it is possible to conduct the operation in stages it is best to start anteriorly, a second and more posterior cut being possible if the first does not produce the desired effect. Thinking on these lines, however, Tow and Lewin suggest that it may be more informative to make the first incision in a definitely selected part of the frontal lobe. A suitable part is the whole of one surface. They have found that the effective orbital leucotomy seems to be at least as good as that of standard leucotomy but with fewer undesirable complications. Their results in a typical but small group of cases compare well with clinical results after the standard operation. There appears to be far less likelihood of any permanent physical complications or of any serious damage to the personality. They regard the operation as worthy of further trial in some cases in which relatively major surgery is thought necessary for the treatment of mental illness. For impulsive and violently disturbed active psychotics, and for patients suffering from chronic affective disorders with great tension and anxiety, it may be the operation of first choice after all non-surgical treatments have failed.

No doubt the argument about the rights and wrongs of leucotomy, both medical and ethical, will go on for a good while, and it is not easy at present to see the outcome. Judgement will certainly be made easier if, as is stated, the more recent modifications interfere less with the essential personality of the patient. Sargant's view is that the patients and their families should be the real judges of the success or failure of the treatment and not the doctor's own theoretical prejudices and emotionally toned judgements. He concludes: "Psychiatry often creates insoluble difficulties for itself when we leave the patient's bedside and become too involved in speculative philosophical issues. If we are only content to remain doctors, consequently searching for new and better practical ways of trying to help our individual patients, the future of leucotomy is assured—at least until better ways are discovered for the practical relief of so much persistent individual human misery and social incapacity." The issue is perhaps not quite as simple as that, but the statement will bear a good deal of careful honest thought.

GAS-GANGRENE INFECTION AT OPERATION.

A MISHAP at the Birmingham Accident Hospital reported by S. Sevitt[1] directs attention to ventilation methods in operating theatres. An open operation on a closed ankle fracture was followed by gas gangrene, and the leg had to be amputated. *Clostridium welchii* was isolated from the infected muscle and from the floor, walls and air of the operating theatre. As the exhaust ventilation of the theatre sucked air and dust from much of the hospital into the theatre and disseminated them there, it was concluded that aerial contamination of the wound had taken place during the operation, and that this had been brought about by the air currents produced by the ventilation.

[1] *Lancet*, November 28, 1953.

Abstracts from Medical Literature.

PHYSIOLOGY.

Artificial Respiration Adapted to Special Conditions.

P. V. KARPOVICH AND C. J. HALE (*J. Appl. Physiol.*, May, 1953) report a study of pulmonary ventilation effected by 10 single-phase artificial respiration manœuvres on 15 conscious non-apnœic subjects. These manœuvres were all applicable under war conditions. It was found that, with the exception of three manœuvres, each could provide adequate pulmonary ventilation in normal lungs. The inferior manœuvres were leg flexion against the abdomen, pressure on one side of the chest and one-arm lift sidewise. The superiority of the double-phase method, combining both active inspiration and expiration, was again demonstrated. The most practical double-phase manœuvre which can be executed under most conditions is the bear-hug arm-lift, which gives 1285 cubic centimetres of tidal air. The intermittent technique for measuring pulmonary ventilation effected by artificial respiration in conscious, non-apnœic subjects gives figures comparable to those obtained on anæsthetized-curarized subjects.

Altitude Stress in Subjects with Impaired Cardio-Respiratory Function.

J. P. MARBARGER *et alii* (*J. Aviation Med.*, August, 1953) report observations on 10 normal young adult men, on eight patients with history and symptoms of *angina pectoris* but without further evidence of acute cardiac disease, and on 14 patients with chronic anæmia whose hæmoglobin concentrations ranged from 5·7 to 12·0 grammes *per centum*. All groups of subjects were taken to simulated altitudes of 10,000 feet (ten minutes' stay) and 18,000 feet (ten minutes' stay for angina patients, twenty minutes' stay for normal and anæmia subjects) in a low-pressure, air-conditioned chamber while resting on a horizontal, low-frequency, critically damped ballistocardiograph. Measurements were made of arterial blood oxygen and carbon dioxide content and cellular components of blood. Direct tonometry of arterial and venous pressures was performed. Simultaneous records were obtained of electrocardiogram, ballistocardiogram, pressure readings and respiratory changes. Analysis of the records and patient behaviour indicate the following facts. Cardiac output promptly increased in all groups of subjects with inhalation of ambient air at reduced barometric pressures existing at 10,000 feet and at 18,000 feet simulated altitude and promptly returned to ground level with inhalation of 100% oxygen. The increased cardiac output occurred as a result of an increase in pulse rate and stroke volume in all groups of subjects. Total effective peripheral resistance to blood flow decreased greatly in all groups of subjects upon exposure to ambient atmosphere at simulated altitudes up to 18,000 feet. "Useful work of the left ventricle" greatly increased in all subjects during exposure to hypoxia. Mean venous pressure remained unchanged in the normal subjects, but increased with duration of stress in both angina and anæmia patients. This was greatest with exposure to severe hypoxia. Electrocardiographic changes in amplitude occurred with hypoxic stress in normal subjects. This included primarily, depression or inversion of *T* wave and depression of *S-T* segment. Similar changes were observed in the anæmia patients and in those with *angina pectoris*, although many of the electrocardiograms of the latter group were abnormal before the test was started. The normal group of subjects and patients with anæmia withstood the test throughout the prescribed course; but the *angina pectoris* group withstood only ten minutes at 18,000 feet before anginal pains occurred in some of the patients, and the test was discontinued at this time period. The over-all responses of the normal and abnormal subjects were so similar that a test to be used for predictive purposes in certain individuals with questionable evidence of cardio-vascular disease (at least the limited type tested by us) could not be achieved. The risk is discussed regarding the transportation of military or civilian patients with impaired cardio-vascular function, as studied herein, by air, either with or without supplemental oxygen to breathe.

Detection and Measurement of Freezing in Tissue.

H. T. MERYMAN AND J. W. MOORE (*J. Appl. Physiol., July, 1953*) state that in the winter of 1950-1951 5000 cases of frostbite occurred among American troops in the Korean campaign. The authors considered methods for the detection of the depth of freezing in tissue in the hope that this might permit an answer to the basic question whether true crystalline freezing occurs clinically in tissue. They selected the method of micro-wave reflection. A conventional radar test unit working on a three-centimetre wave-length was adapted for either laboratory or field use. The authors state that the laboratory study of cold injury is, of course, infinitely simpler than field research, and it is tempting to limit this study to the experimental animal. However, it is always questionable how far one may translate laboratory animal experimentation in terms of human response. If a firm and final answer to this aspect of cold injury in humans is to be achieved, it must come ultimately from the field, and it is with this hope in mind that this relatively adaptable instrumental procedure was developed.

Evaporative Forces, Skin Temperature and Human Perspiration.

C. L. TAYLOR AND K. BUETTNER (*J. Appl. Physiol.*, August, 1953) report that the plotting of skin water losses against average skin temperature (experimental results being used from human exposures to combined conditions of heat, humidity, wind and low air pressure) reveal that significant residual variation is associated with these environmental influences. Since the direction and magnitude of these influences are such as to increase or decrease evaporation from the skin, the phenomenon has been designated as the evaporative effect. This effect cannot be explained in terms of conventional theory, which assumes that a skin temperature change is intermediary to any skin water loss response to changed environmental evaporative influences. The basic evidence for the evaporative effect has been derived from four experimental series variously engaged in by the authors. These sets of data cover the pertinent variables and confirm the generality of the phenomenon. In addition ample corroboration of the wind and humidity influences has been found by reanalysing data from the literature. Explanation for the evaporative effect has been sought in skin water transfers not involving active sweat gland participation. This directs attention to the known processes of diffusion (insensible) losses and skin water storage shifts, and to the much less completely understood processes of penetration, repenetration and osmotic regulation in the human skin. Evidence for each has been analysed, indicating that further investigation of these processes may reveal the mechanism of the evaporative effect.

Venous Return from Skin and Muscles of the Forearm.

I. F. S. MACKAY (*J. Appl. Physiol.*, July, 1953) describes two simple techniques for the measurement of the pressure in a Riva Rocci sphygmomanometer cuff which is necessary to prevent the venous return from the skin and from the muscles of the arm. The pressure necessary for the prevention of the venous return from the skin is approximately five to ten millimetres of mercury below systolic pressure. A lower pressure, approximately 25 millimetres of mercury below systolic pressure, is required to prevent the venous return from the muscles. In order to obtain these measurements the appearance and disappearance of ischæmic pain in the muscles and the skin were used as an indication of the absence or presence of blood flow in the muscles and skin.

Body Composition.

R. M. FORBES *et alii* (*J. Biol. Chem.*, July, 1953) have reported the chemical composition of the body of a normal adult human, forty-six years of age. The whole body contained 19·44% ether extract, 55·13% moisture, 18·62% protein, 5·43% ash, 1·9% calcium and 0·925% phosphorus. On the fat-free basis these percentages are, respectively, 69·38, 23·43, 6·83, 2·4 and 1·16. Analyses are reported on 13 separate tissues and organs and are summed to give the above data. Evidence is presented that the normal adult human body contains approximately 1·8% to 2·5% calcium on the fat-free basis and possesses a calcium:phosphorus ratio of 2:1. The water and fat contents of the adult human body as determined directly are in good agreement with the data obtained by indirect methods.

Unilateral Hypoxia.

H. RAHN AND H. T. BAHNSON (*J. Appl. Physiol.*, August, 1953) report that a method has been described for separation of gas exchange of the two lungs and simultaneous measurement in each of alveolar partial pressures of oxygen and carbon dioxide, oxygen uptake and carbon dioxide output. This method has been used when the left

lung of the dog has been subjected to various mixtures of oxygen in nitrogen. The changes in gas exchange with unilateral hypoxia have been presented and discussed. On the basis of certain assumptions, with the use of data obtained in these experiments and blood dissociation curves for oxygen and carbon dioxide, the blood flow through each lung has been estimated. When the right lung is maintained on 30% strength oxygen and the left lung is made hypoxic, there is a local vasoconstriction in the latter which varies with the degree of unilateral hypoxia. With the left lung breathing nitrogen, 11% of the pulmonary flow goes through this lung in contrast to control values of 40%. If on the other hand the right lung is maintained on air, the hypoxic left lung will constrict less for a given alveolar partial pressure of oxygen.

BIOCHEMISTRY.

Vitamin B_{12}.

C. T. LING AND B. F. CHOW (*J. Biol. Chem.*, May, 1953) have studied the effect of vitamin B_{12} on the levels of sulphhydryl compounds in the blood of rats and of patients with pernicious anæmia. It was found that vitamin B_{12} deficiency in rats, as well as in patients with pernicious anæmia, resulted in a pronounced diminution of the levels of soluble sulphhydryl compounds in blood, which rose again upon the administration of the vitamin. Sulphhydryl groups of plasma proteins were not affected by the deficiency or administration of vitamin B_{12}. The changes in blood sulphhydryl levels were primarily due to those in the concentration of glutathione. Ergothioneine levels in the blood also showed some fluctuation prior to and following vitamin B_{12} therapy, but this change constituted only about 10% of the total values. Folic acid or iron deficiencies did not alter the levels of soluble sulphhydryl compounds in the blood.

J. N. WILLIAMS *et alii* (*J. Biol. Chem.*, June, 1953) have shown that a vitamin B_{12} deficiency in the rat produces a considerable reduction in liver xanthine oxidase and betainehomocysteine transmethylase activities. From a study of the response of these enzymes in vitamin B_{12}-deficient rats to supplementation with the vitamin, it is concluded that the effect on xanthine oxidase was an indirect effect concerned with general protein metabolism. Transmethylase, however, responds completely after a single injection of vitamin B_{12}, which indicates that this enzyme may require the vitamin as a co-factor or co-factor precursor.

Cholesterol.

O. HECHTER *et alii* (*Arch. Biochem.*, September, 1953) have studied the transformation of cholesterol and acetate to adrenal cortical hormones. C^{14}-labelled acetate and labelled cholesterol were perfused through bovine adrenal glands in the presence of ACTH. The two major corticosteroids synthesized by the adrenals, and the cholesterol fractions present in blood and adrenal tissue at the end of the experiment were fractionated and purified. The "free" cholesterol fraction in the adrenal tissue had the highest specific activity of all cholesterol fractions examined independent of whether acetate or cholesterol was employed as the C^{14} precursor. It was observed that the corticoids synthesized from radio-acetate had a higher specific activity than the "free" cholesterol from adrenal tissue, whereas, with C^{14} cholesterol perfusion, the corticoids synthesized had a lower specific activity than the adrenal-free cholesterol.

G. M. TOMKINS *et alii* (*J. Biol. Chem.*, August, 1953) have shown that cholesterol synthesis in the liver is suppressed by steroid administration. Cholestenone, dehydro - iso - androsterone, 7-dehydrocholesterol, and Δ 7-cholestenol were fed to rats, either as a single intubation or for three to seven days, and the rat livers were subsequently examined for their ability to incorporate acetate-C^{14} into cholesterol. The feeding of these cholesterol-like steroids resulted in a pronounced reduction in the recoveries of cholesterol-C^{14} without, however, affecting the values for $C^{14}O_2$.

Iron.

A. R. STEVENS *et alii* (*J. Biol. Chem.*, July, 1953) have determined the loss of radio-iron from the body of the mouse. There was a turnover of body iron of 0·5% per day. Normal animals and animals with increased iron stores regardless of sex showed approximately the same percentage loss of body iron per unit of time.

Uric Acid.

J. B. WYNGAARDEN AND D. STETTIN (*J. Biol. Chem.*, July, 1953) have measured the extent of uricolysis in man by determination of the distribution of isotope in urinary uric acid, urea and ammonia following the intravenous injection of uric acid-1,3-N^{15}. On the basis of isotope recoveries it has been calculated that, of the uric acid administered, some 18% was degraded to other nitrogenous products which appeared in the urine, and about 6% was excreted in the fæces, over a two-week period. From considerations of the size of the miscible pool of uric acid and its turnover rate, it has been concluded that 78% of uric acid was excreted unchanged in the urine. Repetition of this experiment in the same subject, while intestinal bacteriostasis was maintained by means of an orally administered sulphonamide, resulted in no essential alteration in the analytical results procured. Of the administered uric acid 17% was degraded to other nitrogenous products appearing in the urine during the experimental period. These results are taken as proof of the occurrence of uricolysis in normal man and as an indication that the intestinal flora does not make a major contribution to this process.

Molybdenum.

D. A. RICHERT AND W. WESTERFELD (*J. Biol. Chem.*, August, 1953) have isolated from soy flour the dietary factor required for the deposition and maintenance of normal levels of rat intestinal xanthine oxidase, and identified it as a molybdate salt. Sodium molybdate gave saturation levels of intestinal xanthine oxidase when the diet contained less than 0·1 milligramme of molybdenum per kilogram of diet. Purified milk xanthine oxidase contained 0·03% of molybdenum.

Hippuric Acid.

D. SCHACHTER AND J. TAGGART (*J. Biol. Chem.*, August, 1953) have presented evidence that benzoylated coenzyme-*A* represents the activated intermediate in the enzymatic synthesis of hippurate in the kidney. The essential factors in the condensation reaction were found to be limited to benzoyl coenzyme-*A*, glycine and the enzyme. The benzoyl group of benzoyl coenzyme-*A* can be transferred non-enzymatically to cysteine, BAL, glutathione or thioglycollate. While such transfer reactions can interfere with the synthesis of hippurate from benzoyl coenzyme-*A* *in vitro*, their physiological significance is not known at the present time.

Ascorbic Acid.

J. N. WILLIAMS AND A. SREENIVASAN (*J. Biol. Chem.*, August, 1953) have presented evidence to indicate that ascorbic acid functions in two places in the tyrosine oxidase system, one early in the scheme and the other in the conversion of homogentisic acid to aceto-acetate and fumarate. In addition to ascorbic acid, glutathione and labile factor, for which a low concentration of 2,6-dichlorophenol indophenol can substitute, appear to be additional co-factors required by the system.

Pantothenic Acid.

H. P. KLEIN AND F. LIPMANN (*J. Biol. Chem.*, July, 1953) have reported finding a parallel between coenzyme-*A* levels and the synthesis of cholesterol, as well as of total lipides, in the liver of pantothenic acid-deficient rats. A depression of steroid and fatty acid synthesis was observed in normal liver slices in the presence of the antimetabolite pantoyl-p-anisidide. This interference appears to depend on a relatively low content in coenzyme *A*. A partial reversal could be obtained with pantethine, but not with pantothenic acid.

Œstradiol.

D. D. HAGERMAN AND C. A. VILLEE (*J. Biol. Chem.*, July, 1953) have shown that œstradiol increases the oxygen consumption of human endometrium *in vitro* and shifts the metabolism of glucose and pyruvic acid toward more complete oxidation. The data are consistent with the hypothesis that œstradiol exerts this effect *in vitro* at some point in the Krebs tricarboxylic acid cycle.

Chloramphenicol.

M. E. SWENDSEID *et alii* (*J. Biol. Chem.*, April, 1953) have shown that the feeding of chloramphenicol to rats results in greatly reduced levels of liver xanthine oxidase, whereas the activity of intestinal xanthine oxidase remains normal. By dialysis experiments it was indicated that this effect was due to a diffusible inhibitor which was not chloramphenicol itself.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XCIII.

SYCOSIS BARBÆ (COCCOGENIC SYCOSIS).

SYCOSIS BARBÆ, a staphylococcal infection of the pilo-sebaceous follicles of the beard area, appears to be less common now than it was in the pre-antibiotic era. However, whenever it occurs it must be treated as a serious skin disorder, often chronic in its course and causing physical and psychological damage to the patient. The condition was formerly referred to as "barber's itch", but it is doubtful whether many cases are acquired today from the barber's shop; nor does the word "itch" adequately describe the sensation of soreness and the discomfort associated with a full-scale attack of *sycosis barbæ*.

Once established in and around the hair follicles of the beard area, the infection becomes a major problem. Its cure demands a concentrated and balanced effort in which the services of general practitioner, dermatologist, pathologist and ear, nose and throat specialist may all be needed.

Today it is possible to give an excellent prognosis in nearly all cases. This is particularly so when the organism has been isolated and its antibiotic sensitivities have been ascertained. Epilation of the beard area with X rays is now rarely necessary.

Ætiology.

The majority of patients are males over the age of puberty. Predisposing causes and associated conditions include the following:

1. Infection of the nose and/or the paranasal sinuses with pyogenic staphylococci. Such an infection continually implants infected material on the upper lip and adjacent parts of the face.

2. The seborrhœic state. Patients with seborrhœic dermatitis or severe seborrhœa are particularly susceptible to this (and other follicular) infection.

3. Other infective states. Furunculosis, *otitis externa*, blepharitis and conjunctivitis may be present and provide a nidus of staphylococcal infection. Impetigo of the beard region may be a forerunner of *sycosis barbæ*.

4. Trauma. Repeated minor trauma—for example, that produced by a blunt razor or shaving too closely—undoubtedly facilitates entry of the organisms into the hair follicles.

5. Occupational factors. The abrasive effect of dusts and the well-known tendency for oils and greases to irritate the follicular apparatus of the skin are examples of occupational influences in ætiology.

6. Emotional factors. The compulsive habit of squeezing and picking skin blemishes exhibited by some persons may be the starting point of sycosis.

The infecting organism is a *Staphylococcus pyogenes*, a coagulase-positive strain of *Staphylococcus aureus*. This organism can be demonstrated in virtually all cases of *sycosis barbæ*. It is a known pathogenic inhabitant of the upper part of the respiratory tract, particularly of the nose. It is estimated that as many as one-third of the population are nasal carriers of *Staphylococcus aureus*. Often it is possible to correlate the serological type of *Staphylococcus pyogenes* occurring in *sycosis barbæ* with that occurring in the nose, eyes or ears of the same person. Persistence of the organism in such foci explains the characteristic tendency of the disease to relapse. Nasal discharges, contaminated handkerchiefs and hands may reestablish an infection.

Pathology.

The pathology is admirably summarized by Ormsby and Montgomery in their text-book "Diseases of the Skin" (reprinted October, 1951). They make the following statement:

> The essential process is an indolent type of folliculitis and perifolliculitis caused by microorganisms. There occurs primarily a collection of leukocytes and serum in the mouth of the hair-follicle. This staphylococcus abscess then extends downward in the follicle and to the mouth of the sebaceous glands and now occasions a perifolliculitis consisting of edema and cellular infiltration of leukocytes, plasma-cells and proliferating connective-tissue cells, a process which may involve the entire cutis. The walls of the hair-follicle are edematous and permeated by wandering leukocytes, as is the neighboring epidermis, the latter becoming slightly acanthotic. The suppurative process often destroys the hair-follicle, the latter becoming replaced by scar tissue. Commonly, however, the hair papillæ escape destruction, so that permanent alopecia may not result. In lupoid sycosis all the structures about the hair, including the hair papilla, are destroyed, thus leaving a permanent scar.

The dermal inflammation in chronic sycosis is associated with masses of infiltrating lymphoid cells, in which are a high percentage of plasma cells and numerous foreign body giant cells.

Clinical Manifestations.

The disease may be acute in its onset, or it may exist for months as a smouldering and localized folliculitis of the upper lip or chin upon which a severe attack may develop. Early lesions are uncomfortable, discrete, red, follicular papules, which mostly become yellow pustules pierced by hairs. In the early stages the hairs are firmly held in their follicles, but after several days can be extracted fairly easily, the gelatinous root sheaths being brought with them. Burning and itching occur, and when the process is extensive, actual soreness and tenderness are present.

The pustular eruption tends to become symmetrical and occurs on the face, lips and chin. Chronic cases may involve also the eyelashes, eyebrows, scalp, axillæ and pubes; but such wide distribution is not common.

In severe cases the lesions are crowded together, the affected skin being studded over with red papules, yellow pustules and loosely attached yellow crusts. Extension of the infection deeply into the dermis may cause tumefaction and larger perifollicular abscesses. Fresh crops of the elementary lesions may be mixed with healing pustules, subsiding inflammation and possibly small follicular scars. Hairs grow more sparsely as the infection lingers.

In lupoid sycosis the process is more destructive and spreads peripherally, causing progressive destruction of hair follicles, associated with atrophic scarring.

Diagnosis.

Clinically, diagnosis is not difficult. An essential step is to identify the infecting organism and determine its sensitivity to antibiotics. When possible, cultures should be made from the nose as well as from the pustules.

Tinea barbæ can often be distinguished clinically by the appearance of a large, often single, brawny red granulomatous lesion with loosened hairs lying irregularly on its surface, the whole effect being one of doughy inflammation and bogginess. Discovery of the causative fungus in cultures confirms the diagnosis.

Impetigo contagiosa may be due to the same staphylococci or to a mixture of pyogenic staphylococci and hæmolytic streptococci. Yellow-crusted, asymmetrical areas of infection arise on an inflamed surface, there being no particular follicular tendency. Pustules and bullæ may occur. It is a much more superficial infection and occurs mainly in children and teen-agers. It can, however, precede sycosis in adult males, hence the importance of early treatment.

Pustular acne will show somewhere the fundamental comedones plugging the pilo-sebaceous orifices. With these will be seen scattered red papules, pustules and possibly inflamed cysts. The seborrhœic diathesis may be obvious—a greasy skin and scalp, dandruff, seborrhœic dermatitis. Acne lesions may be present on the back and chest. The pustular contents of acne lesions are often sterile in spite of the quantity present. *Staphylococcus albus*, an organism of doubtful pathogenicity, may grow on the media.

Drug eruptions due to bromides and iodides can closely simulate *sycosis barbæ*. Bromides may cause a pustular eruption, acneiform in nature and occurring most commonly on the face. Large granulomatous nodular lesions may be seen. These large lesions may persist for weeks after withdrawal of the drug. Iodide eruptions may be erythematous, papular and pustular, and in this form may cause confusion in diagnosis. Granulomatous lesions, bullæ and petechiæ may be associated and symptoms of iodism may be detected.

Course and Prognosis.

In early cases the condition will respond well and rapidly to appropriate antibiotic and local therapy. In chronic and neglected cases it may linger indefinitely and cause much scarring and psychomorbidity. The tendency to chronicity and to relapse during treatment should focus attention on the nasal mucosa and other areas of possible reinfection. It may mean also that the organisms have become "antibiotic-fast", and that a new approach is needed. Contact with abrasive dusts or with oils and greases may prolong the illness. The seborrhœic person already has a tendency to acquire follicular lesions and his case must receive particular attention. In the average case the condition will clear up within three weeks and should remain cured. At times permanent cures can be obtained in as little as one week.

Treatment.

1. The appropriate antibiotic should be administered by injection or by mouth. Many of the infecting staphylococci have acquired resistance to penicillin, and it is not unusual to find sensitivity to all the antibiotics except penicillin. Erythromycin is probably the most commonly effective antibiotic as revealed by sensitivity tests and supported by clinical observations. Terramycin and aureomycin are also very effective. The oral administration of these agents should be "covered" by large doses of vitamin *B* group (two or three tablets three times daily). Under-dosage with antibiotics can be more disadvantageous than over-dosage, and a suggested course of treatment with erythromycin, terramycin or aureomycin is the administration of one to two capsules four times daily (six hourly), continued until the condition has cleared or until attempted culture produces no growth. When penicillin is indicated by sensitivity tests, it should be given in doses of 600,000 units daily, half as procaine penicillin with 2% aluminium monostearate solution and half as the sodium salt of penicillin G.

2. By way of local therapy, the appropriate antibiotic applied in saline solution or in ointment form is again the treatment of choice. A 3% aureomycin ointment is excellent. Erythromycin, five milligrammes in one gramme of "Vaseline", is even more effective. Bacitracin ointment is also of use. The combination of neomycin and bacitracin as "Neotracin" ointment is more effective than either alone. Penicillin ointment sometimes has a high sensitizing index and is not recommended for general use. Erythromycin and aureomycin used locally seem to have the least sensitizing effect on the skin. The local use of sulphonamides is unnecessary and potentially dangerous.

Local treatment should include application of the ointment inside the nostrils, and to the eyelids and ears if these are involved. It should be continued for at least two weeks after apparent cure.

A paint of 10% aqueous ichthyol solution applied twice daily is useful if sensitivity to a previous application has occurred. Gentian violet in 1% to 2% aqueous solution is also needed at times. It is of particular use when the antibiotics are not tolerated locally. "Vioform" (3%), in solution or in zinc paste, is useful but messy. It also has a rather high sensitizing index. "Quinolor" ointment has been a trusty stand-by; but I regard it and the other agents mentioned above as inferior to erythromycin or aureomycin ointment for local treatment. Stock and autogenous staphylococcal vaccines may occasionally be useful in the now rarely seen chronic cases.

X-ray epilation of the affected areas is seldom needed today. It is a procedure to be avoided if possible, and I have not heard of its being used in Sydney since the war ended in 1945. Accompanying conditions such as seborrhœic states must receive concomitant treatment. Reduction of fat and carbohydrate in the diet and correction of any obvious defects (bad teeth, infected tonsils *et cetera*) are combined with local treatment. Sulphur (2%) and salicylic acid in a water washable ointment base are applied to the scalp frequently and washed out with soap and water. "Pragmatar" and "Eskamel" are recognized anti-seborrhœic agents and can be used freely under supervision.

In cases in which much œdema and serous exudation are present, one can often obtain a history of some injudicious local application. Such applications include sulphanilamide (and other sulphonamide) ointment. Antihistaminic agents in cream form are not well tolerated and should not be used in this or any other inflammatory dermatosis. Their sensitizing index is high. Their use should be restricted in general to such conditions as localized pruritus and insect bites.

The exudative type of condition will initially require treatment with lotions such as Burow's lotion or boric lotion (0·5% to 1%) applied frequently as wet packs until most of the exudation has ceased.

It is advisable for the patient to continue to shave daily or every second day. Complete cessation of shaving may lead to thick crusting on the surface and matting of the hair. Shaving may be best performed with an electric razor, although this device is not suited to some patients. When this is so, a brushless shaving cream may be preferred. When a shaving brush and razor are used, care must be taken to ensure that they are sterilized before and after use. This is readily done by immersion of the razor and the last half-inch to one inch of the bristles of the brush in boiling water for thirty seconds before use. After use they are again sterilized in a similar fashion and stored until next needed in a 2% aqueous solution of "Dettol" or "Zephiran" (1 in 1000). This is washed out and the boiling procedure is repeated before the next shave. After shaving, the face is splashed with warm water and patted dry, and an astringent lotion of 0·5% zinc and copper sulphate solution in camphor water is dabbed on. The selected local agent is then applied, and its application is repeated twice in the twenty-four hours.

X-ray therapy in small, non-epilating doses—for example, 75*r*—can be given at intervals of seven to ten days and is of undoubted assistance when so used. It has a direct effect in reducing the inflammatory reaction and an indirect effect on the bacterial infection.

Conclusion.

Staphylococcal *sycosis barbæ* is less commonly encountered now than before the introduction of antibiotics. Its occurrence may be associated with the presence in the upper part of the respiratory tract of serologically identical organisms. The sequelæ of scarring of the skin and the mind can usually be avoided by a properly balanced regime of antibiotic therapy.

REX BECKE,
Sydney.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 27, 1953, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. A. J. MURRAY, the President, in the chair.

Heart Disease in Industry.

DR. F. A. E. LAWES read a paper entitled "Heart Disease in Industry" (see page 233).

DR. U. L. BROWN read a paper entitled "Heart Disease in Industry" (see page 236).

In opening the discussion, DR. W. L. CALOV said that it was a common experience to find that one's admiration for a paper was conditioned by the similarity of the speaker's views to one's own. He said that he admired the papers read by Dr. Lawes and Dr. Brown; but for the sake of debate, he was happy to say that he disagreed with some of the views that had been expressed. Dr. Calov went on to say that he thought that increase in the knowledge of coronary occlusion had been hampered by the acceptance of the term "coronary thrombosis". It was true that at post-mortem examinations of persons dying of myocardial infarction a thrombus was frequently found obstructing a coronary artery; but was it known that the thrombus was the cause of the occlusion? Was it not possible that an occlusion had been caused by some other means and that the thrombus had formed afterwards? Of course it could not be denied that a clot could form on the diseased lining of an artery; but the formation of a clot was a gradual process. Dr. Calov asked those present to consider the case of a man who had had no symptoms of cardiac disease, but who suddenly became stricken with the symptoms of coronary occlusion. Was it believable that a clot was being gradually built up without causing symptoms and then was suddenly built up further so as to block the artery and cause terrible symptoms? Dr. Calov did not believe this. Dr. Calov asked those present to recall the moving picture of a clot *in vitro* shown by Professor Best on his recent visit

to Sydney. The clot was in a glass tube through which blood was flowing. The clot was seen to move, and portions were seen to separate from it. Was it not possible that such things occurred *in vivo*? And was it not possible that such movement of a clot was more likely when the blood pressure was elevated and the velocity of the flow of blood was increased? An argument against thrombosis as a necessary cause of obstruction was the finding of Wright Smith, of Melbourne, that in persons who died suddenly of coronary disease, thrombosis was seldom found. Dr. Lawes had said that the theory of obstruction of a coronary artery by an atheromatous plaque had now been discarded. Dr. Calov pointed out that it was not uncommon to find a diseased coronary artery obstructed by debris. Whether this debris had blocked the vessel by a natural process or whether it had been dislodged by the post-mortem operation he could not say. Lewis, in his book on peripheral vascular disease, stated that atheromatous material could be discharged into the lumen of a vessel and be swept on in the current of blood to cause obstruction further towards the periphery. If such a thing could happen in a vessel in a limb, why not in a coronary vessel? Dr. Lawes had mentioned King's paper on subintimal hæmorrhage. King had attempted to prove on grounds of pure dynamics that these hæmorrhages from subintimal vessels could not cause bulging of the intima sufficient to cause obstruction to the lumen of the main vessel. Dr. Calov had no doubt that it could be proven one way or another by pure dynamics provided one had all the data. But he did not believe that all the data were known. It was one thing to prove that the pressure in a metal pipe was at such-and-such a level; but it was quite another thing to prove it in pipes of vital tissue—and diseased tissue at that—in which there were variables that were not understood. Dr. Calov suggested further that the pressure in a subintimal vessel might depend on the pressure within the artery at a point proximal to the site of obstruction and that the lumen of the artery at the site of origin of the subintimal vessel might be considerably wider than at the site of obstruction. Dr. Lawes had said that if a man got cardiac pain as the result of exertion he would stop what he was doing. Dr. Calov said that the man would stop if he knew the cause of the pain. But he might not realize that the pain was cardiac. On the subject of pain, he was glad that Dr. Brown had drawn attention to the fact that the pain of coronary disease did not necessarily have the classical pattern. Dr. Calov remembered well a patient who was subject to pain that commenced in the little fingers and gradually came up the arms as the man walked. Later in the man's life the pain spread across his chest. At first that man had pain only on walking to the railway station on his way to work. He was able to do his work and to play cricket on Saturday without discomfort. Dr. Brown had suggested that in such cases freedom from pain later in the day might be due to a freer flow of blood through the coronary circulation as a result of exertion. Dr. Calov thought that the reason might be threefold. In the first place the man got the pain after breakfast, and it was known that angina was more likely to occur after a full meal. In the second place, the erect posture had been assumed for only a short time when the pain had commenced, and much of the blood might have been in the splanchnic vessels as the result of gravity. In the third place there was the man's disinclination to go to work. Dr. Lawes had mentioned the occurrence of coronary occlusion in a man affected with pulmonary fibrosis. Dr. Calov suggested that a man affected with pulmonary fibrosis and emphysema and resulting anoxia would be more likely to die of a coronary occlusion than if he were not so affected. Dr. Calov pointed out also that a man might be very breathless and cyanotic from pulmonary fibrosis and emphysema, without presenting objective evidence of right-sided heart failure; in many such cases the cardiac shadow appeared to be quite normal on fluoroscopic examination. Dr. Calov said that he was glad that cardiac neurosis had been mentioned. He stressed the importance of recognizing that a cardiac neurosis could and frequently did exist in the presence of organic cardiac disease. He was disappointed that neither Dr. Lawes nor Dr. Brown had discussed valvular disease. He recalled cases of advanced mitral stenosis in which the patients had lived a full and happy life and had performed athletic feats demanding great strength and stamina. Dr. Calov suggested that, if these people had been denied the right to lead the lives they had led, they might have been soft, weak persons affected with cardiac neurosis as well as mitral valvular disease. Dr. Calov concluded by saying that he had enjoyed a particularly good dinner that night, and he had been warned that by the time the discussion commenced he would be asleep. Dr. Lawes and Dr. Brown could take it as a compliment that he had remained awake and was then in the same state of consciousness as he was usually in at that time of night.

DR. D. A. W. DOWNIE said that he had one problem to raise, and that was whether effort had any effect at all on *angina pectoris*. He himself believed that effort had no more effect on it than in its production of pain in the skeletal muscles, and that it did not lead to fibrosis. Dr. Downie said that he was a medical officer dealing with coalminers; insurance for about 15,000 men was involved, and the incidence of heart disease was about one case or one follow-up per week. If any deduction was to be drawn from the difference between those figures and what Dr. Brown had said about the railways, it might seem that the coalminer's laborious occupation prevented him from getting heart disease. Dr. Downie did not think that effort bore any relationship to coronary occlusion. The question of pressure in the *vasa vasorum* had been raised. Dr. Downie said that thrombosis was more likely to occur during sleep, or where there was sluggish blood flow, than during activity. He finally referred to an article in *The Lancet*, in which Sir Adolphe Abrahams had referred to one of his patients who was the champion middle distance runner in the British Isles; he had mitral stenosis.

DR. E. H. STOKES said that he had appreciated Dr. Lawes's paper and Dr. Brown's humanitarian attitude in connexion with coronary disease. Dr. Lawes had said that murmurs were sometimes too much stressed. Dr. Stokes said that one frequently saw the bad results of that attitude in clinical practice, the patient often being a young boy who had been forbidden to take part in exercise. Referring to cardiac neurosis, Dr. Stokes said that the pain was likely to be situated in the mammary region rather than in the retrosternal region. The nervous factor had to be taken into account in the assessment of cardiac function. It was well known that sufferers from angina got attacks of pain when walking; Dr. Stokes believed that the pain did have an effect upon the progress of the angina. Dr. Stokes said that he was not going to attempt to answer the questions put forward by Dr. Calov on the subject of coronary thrombosis and coronary occlusion. But the judges at the Workers' Compensation Court had a very simple method of deciding whether those conditions were the result of effort in a given case: if the effort had occurred just before the man's death, an award was granted. The unfortunate part of that method was that industry had to pay for the whole of the man's preceding condition as well as his coronary occlusion.

DR. W. J. MCCRISTAL, on a personal note, said that he did not quite like Dr. Brown's analogy of the occurrence of coronary occlusion and grey hairs, especially as he had predilections for picking a certain somatotype as a more usual candidate for coronary occlusion. Speaking more seriously he said that it was rather upsetting to find the variability of viewpoint as to incidence of intimal hæmorrhage in coronary occlusion. When pursuing abroad the subject of anticoagulant therapy he had spoken to Dr. Wartman in Chicago in 1950, who, with others, found intimal hæmorrhage in 60% of 400 cases of occlusion. Since Dr. McCristal regarded this feature as the pivot for the integration of coronary syndromes, he felt that it was necessary to settle the issue. He said that it was quite in accord with vascular physiology to correlate anginal pain with inactivity subsequent to work. He instanced the case of a man whom he had seen recently with left bundle branch block who worked comfortably all day sewing soles on blucher boots and yet had anginal pain going home in the bus. Such a man was more likely to die in his sleep than at work. Dr. McCristal said finally that the initiated should instruct their less experienced colleagues as to terms; for example, coronary insufficiency with effort in a hypertensive heart had quite different prognostic and therapeutic implications compared to coronary insufficiency or failure in an atherosclerotic heart. In the latter instance death might occur quite dramatically when the imbalance between coronary supply and cardiac demands was further exaggerated in an ill-nourished heart.

DR. W. E. FISHER said that it was perhaps a suitable occasion on which to revive an old suggestion going back to the days of Allbutt. The most vexed problem was that of the ætiological connexion of myocardial infarction with effort. In their papers, Dr. Lawes had reviewed a wide range of opinions, indicating here and there his personal inclinations, Dr. Brown had given a refreshingly assured account based on personal experience, but had refrained from that particular problem, and Dr. Calov had meditated, as all might do, but in public for the meeting's benefit rather than at his own fireside. Neither Dr. Lawes, as a result of an extensive review, nor Dr. Brown, from first-hand

experience of many patients, nor Dr. Calov, despite the freer rein he allowed himself, was prepared to commit himself definitely on the ætiology of acute infarction in anatomical and physiological terms. So it was that members of the medical profession could be found lined up on one side or the other in court. This did not do them much credit in lay eyes. In the first stage of Allbutt's career in Leeds, he had been able to persuade the Benches to abandon that method; at his suggestion cases were discussed by doctors away from the court, and their final decision was handed to the judges unanimously or with a minority opinion. When Allbutt left Leeds he had been congratulated on the results achieved. Dr. Fisher went on to say that years earlier he had had something to do with workers' compensation work, and he had put the suggestion to the largest insurance company in Sydney; they were not interested. He thought that such a procedure would be worth while and in no type of case more so than that under discussion.

Dr. Murray, from the chair, said that he had been interested in the remark of Dr. Downie that coronary occlusion occurred more commonly during sleep than at work. He remembered that it seemed to have been that way during his general practice days. He asked whether there were any figures on the point.

Dr. Lawes, in reply to Dr. Murray's question, said that he did not know the exact figures. Master had stated that only about 2% of coronary occlusions occurred during activity; the remainder occurred at rest. During sleep the circulation was considerably slower than during the day. Dr. Lawes said that he agreed with Dr. Calov on most points; disagreements depended entirely on how one looked at things, on whether one took a mechanistic view of the subject or not. He himself did not believe that coronary occlusions occurred as a result of effort; but one could conceive of the occurrence of infarction of muscle or even death of muscle from prolonged anoxia. That was why he had always told the courts that if a man died during effort or a few minutes afterwards, a causal connexion must be admitted. Therefore he agreed with Dr. Calov that sudden death might occur as a result of effort. Dr. Calov had mentioned anoxia resulting from pulmonary fibrosis; it had to be admitted that if that condition was present the heart would be embarrassed, but sometimes there was no evidence in a man's history that anoxia had occurred. Dr. Lawes said that Dr. Calov's remarks about cardiac neurosis occurring at the same time as organic heart disease had raised a most important point. Heart patients were often greatly afflicted with neurosis, and many a man with heart disease could be returned to work if he was given a helping hand and some encouragement. Dr. McCristal had raised the question of what coronary insufficiency was. The condition was one in which the heart was called on for an effort that it was unable to make because of insufficient blood. There were many degrees, even leading to death. Dr. Lawes welcomed Dr. Fisher's suggesion, and said that it would overcome many difficulties. Dr. Lawes said also that he had often spoken to Dr. Calov unofficially about these matters, but had always intended to meet him and discuss them in greater detail.

Dr. Brown, in reply to Dr. Downie's question as to whether or not an anginal seizure had any continuing or lasting effect on the myocardium, said that so far as he could tell from his reading and from evidence given in the courts, it was fair to say that the pain of angina, in passing, left the myocardium in exactly the same condition as before. Dr. Brown said that he was attracted by Dr. Fisher's suggestion that a conference of doctors should discuss the medical aspects of claims heard in our courts and that the opinion of such a conference be accepted by the tribunal as the medical evidence in the case, rather than that doctors be called to give evidence by the parties to the dispute. He was surprised to learn that it had been rejected years ago, for it was his opinion that this could be a more reasonable way to determine whether an effort at work or on the periodic journey had any relationship to a heart attack. Doctors, as yet, could not positively answer all the questions in their own minds. They could consider the post-mortem appearances and try to explain them in terms of related knowledge and function, but there remained much conjecture on some aspects of the subject, and that was mainly why such cases reached the courts and such bizarre medical evidence was given.

Dr. Brown said that his own problem was that of maintaining a man in employment and adapting the job to the man's capacity for comfortable effort. It was to be expected that heart attacks and possibly sudden death would occur in such cases; and if these occurrences had a time relationship to the work, it seemed to be the rule that the man's union alleged a causal relationship and the employer was put to the expense of defending the claim. Dr. Brown felt that if the medical aspects of such claims were determined by a conference of selected doctors, he would feel much freer in continuing such men in employment, subject, of course, to the man's own desires and providing he could get to and from the job comfortably, for there was no doubt that most men must continue in employment long after the manifestation of coronary disease.

In answer to the President's question that from his former experience in general practice his impression was that acute coronary occlusion occurred more at night, Dr. Brown said that the question often arose in the courts, and statistics were quoted to support one or other view. Perhaps the correct answer was that acute coronary occlusion would occur in any case when the natural progression of the disease deprived an area of myocardium of its blood supply and no alternative blood supply offered, and whether this occurred during the day or night was coincidental. In other words, it was a purely temporal relationship. If, however, by "day and night" was really meant time when active or at rest, it was his impression that more attacks of acute coronary occlusion became manifest under or following conditions of rest because an adequate blood supply through narrowed, branching arteries was more likely to be maintained if the blood was flowing with the greater velocity and head of pressure that accompanied effort.

Referring to the phrase "subintimal hæmorrhage", Dr. Brown said that Patterson had used it in 1936 when he was reporting on the microscopic appearances of coronary atheroma. Very soon pathologists, the world over, were reporting on these lesions, and lively discussion continued as to their true nature. Patterson had suggested that they were due to hæmorrhage from ruptured *vasa vasorum* and offered the view that they conceivably might contribute to the cause of a coronary occlusion by forming a lake of blood which, in expanding, lifted up the intima, causing it to project into the lumen of the coronary artery and thus obstructing partially or completely the blood flow through the artery. Those favouring the view that coronary occlusion was related to effort immediately seized on Patterson's "subintimal hæmorrhages" to support their view and said, in effect, that the raised blood pressure of effort contrasting with that at rest caused rupture of the *vasa vasorum*, forming a pool of blood which, in expanding, blocked the artery, and this view was put forward in the courts in the attempt to relate a particular occlusion to either a single effort at work or the general efforts made by a man during his working day.

Dr. Brown said that his own opinion was that these so-called subintimal hæmorrhages were not hæmorrhages at all, but hæmorrhagic lesions, and were part of the degenerative process of atheroma, and that the red cells had escaped from the lumina of the *vasa vasorum* by diapedesis through the unbroken wall. Despite wide reading on the subject, he knew of no instance where a pathologist had demonstrated a rupture. He found it impossible to accept that blood escaping from a possibly ruptured vas whose pressure was not much more than zero millimetres of mercury could expand in tissues where the surrounding pressure was much greater—namely, something less than the diastolic pressure of the patient, a problematical range of 20 to 80 millimetres of mercury, and greater still in accompanying hypertension.

This impossibility, of course, was obvious in the case in which the affected *vasa vasorum* were the terminal twigs of coronary branches far removed from the point of origin of the branches. It was perhaps less obvious, but none the less true, in the case of the luminal capillaries which had a short course in the wall of the artery from which they arose; this aspect had been clearly reviewed by Edgar King (in *Australasian Annals of Medicine*, May, 1952), who concluded that cardiac infarction was not due to vascular obstruction from subintimal hæmorrhages of coronary vessels.

Replying to the question that subintimal hæmorrhages were possibly post-mortem or agonal phenomena, Dr. Brown said that the evidence was against this possibility, as in those cases of coronary atheroma in which multiple hæmorrhagic lesions occurred a wide range of age changes were apparent.

Dr. Murray, from the chair, said that those present were grateful to the speakers for the admirable way in which the subject had been covered. He had been most impressed with Dr. Brown's management of men who had had coronary occlusions. His common-sense approach and his message of good cheer to the sufferer should not be confined to industry. It would be well if the method was more widely

followed in other branches of medical practice. Dr. Murray said that he would like to see Dr. Fisher's (or Sir Clifford Allbutt's) suggestion put into operation. They would rely on Dr. Fisher to push it on now that the matter had been raised.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE TRANSPORT OF A WOMAN TO THE PARRAMATTA HOSPITAL.[1]

Colonial Secretary's Office,
Sydney,
9 March, 1846.

Patrick Hill, Esqr.,
Colonial Surgeon,
Parramatta.

Sir,

In acknowledging the receipt of your letter of the 5th instant enclosing a statement by Mr. Chas. Macarthur of Ellerslie of the circumstances under which the woman named in the margin (Catherine Donnelly) was forwarded to the Parramatta Hospital, I have the honor by direction of His Excellency the Governor to remark that he does not see that Mr. Macarthur's explanation removes the difficulties in the case. The real point in the case involves the question whether it can be permitted to Gentlemen in any part of the country to put a helpless Invalid on top of a Wool Dray with directions that he or she shall be dropped at a Government Hospital two hundred or three hundred miles distant. The matter is not so much a question of expense (though this is of some importance) as one of common charity.

I have, etc.,
E. DEAS THOMSON.

Special Correspondence.

PARIS LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Artificial Hibernation.

A NEW treatment against shock of any origin (traumatic shock, post-operative shock and shock due to infection) has been studied for the past few years in army and civil hospitals in Paris. It is now used in several other cities of Europe. Dr. H. Laborit, a navy surgeon and physiologist, who is the chief author of the method, called it "artificial hibernation". The principle is completely new and based on a special conception of the organism's responses to aggression.

Basic Principles of the Method.

The responses to aggression have in fact one goal—the maintenance of homœostasis in all its aspects and thus of the usual conditions of the life of the tissues. This homœostasis is considered by Laborit as the expression of the superior being's freedom from environment. The classic therapy helps and stimulates the organism fighting for its homœostasis. But the possibilities of the thus stimulated organism can be overstepped because of the gravity of the aggression or because of the organism's own weakness. In such cases the shock becomes incurable. The result is that defence of homœostasis at any price ends in exhaustion and death. Is there really no other possible solution?

Dr. Laborit, after many experiments on animals, reached the following conclusions. It is possible by means of certain drugs to force a non-hibernating mammal to abandon its "proud homœostasis" without any fight or reaction. At the same time it is possible to reduce the "standard of living" of its cells. Then this organism enters a "slowed-down" state of life which can be compared with that of a hibernating mammal. It is "submission to environment". This can be accomplished by the physiologist without danger to the animal's life, and the subject is later able to recover its homœostasis and its free, normal life. In the state of hibernation all the organism's needs are slight, its responses to aggression are very weak, and it is nearly impossible to provoke a shock. Therefore, every time the organism has to face a very serious aggression which will perhaps exceed its defence possibilities, the responses must be neither helped nor stimulated. It has been shown that such responses endanger life in this condition. On the contrary, these responses must be stopped, the organism must be slowed down to the state of hibernation, since in this state the consequences of aggression are much less.

Application of the Method.

The problem lies in paralysing the organism's responses and in reducing its metabolisms. These two effects must, of course, be kept under constant and perfect control. When required the hibernated organism has to be brought back easily to its previous homœostatic state. Without going into complete technical details, which would be too long for the scope of this letter, the general method can be summarized as follows.

1. Inhibition of the organism's responses is obtained by means of a "lytic cocktail". This "lytic cocktail" is made by association of ganglioparalysers and vagolytic and antihistaminic drugs. These drugs have a "potentializing" effect—that is, each drug augments the individual effect of the others—and so only very small doses are necessary and the "cocktail" is only slightly toxic. The general effect is a neuroplegia, a paralysis of the autonomic nervous system and also a certain reduction of the metabolism. This general effect can be used alone, without the addition of cold. In that case it is only a "disconnexion" and not a true hibernation.

2. To produce reduction of the metabolism, general refrigeration is used. It is very easy because of the neuroplegia and because of the complete absence of resistance. A few rubber bags of ice are enough. In fact the temperature begins to fall merely as a result of the "lytic cocktail". The drop occurs without any reaction and—most important—without any shivering.

The Patient in the State of Hibernation.

The patient under hibernation is half-conscious or, more often, completely unconscious. He is given every day about four pints of liquid, with salt and sugar, by intravenous perfusion. The rectal temperature must be kept between 91° and 95° F. The pulse is slow, the blood pressure has come down by three or four points. Arrhythmia, if any, is usually improved. Breathing is deep and slow. The complexion is pale. The masseters are relaxed. The pupils are in miosis. Analgesia is complete. Shivering must never occur. Basal metabolism is reduced by 10% to 20%. Urine is in relation with the quantity of intravenous liquid, and its chemical composition is normal. Extracellular fluid has a normal composition. Azotæmia is low. Alkaline reserve is normal. The observation of the patient has to be supervised by an anæsthetist well trained in the method. This state of artificial hibernation can last for several days.

The "Warming-up" of the Patient.

The "warming-up", when decided upon, is accomplished by reduction in the doses of drugs and removal of ice. The return to normal temperature must be slow and progressive. It is a delicate period in the treatment. When there is any strong reaction and especially when there is high fever, it is sometimes necessary to "hibernate" the patient again for a short time. The "warming-up" can be helped by ascorbic acid, cortical hormones, "Prostigmin", androgenic hormones and blood transfusions.

Indications and Results of the Method.

The first indication today seems to be the prevention and treatment of surgical shock. Hibernation is begun before the operation. A very small dose of anæsthetic is sufficient during the operation; hæmorrhage is reduced. Hibernation is continued during the first few days of the post-operative period. The method is used only for very severe operations, and in all cases in which grave shock is to be feared. It has been used above all when operation was considered impossible with the usual method of reanimation. In very serious traumatic shock hibernation has given notable success. Army surgeons in Indo-China have used the method to transport severely wounded soldiers without pain or shock under difficult jungle conditions. Patients with malignant hyperthermia after operation and in neurosurgery, obstetric

[1] From the original in the Mitchell Library, Sydney.

shock *et cetera* have been treated, and very favourable reports published. In medical science hibernation has shown an interesting effect in cases of severe infection.

Today treatment by hibernation is still in its experimental stage. The technique and indications of the method, the physiological behaviour of patients under hibernation, the consequences on the organism are being studied and are becoming better known. However, it is still a difficult and delicate method to be used only by specialists. But it has given good results when all seemed lost, and made possible operations which could not otherwise have been attempted. Therefore it seems reasonable to found some real hopes on the future of "artificial hibernation".

Correspondence.

THE TREATMENT OF ACUTE POLIOMYELITIS.

SIR: The articles by Dr. Gordon Colvin and Dr. S. E. J. Robertson on poliomyelitis (M. J. AUSTRALIA, January 16, 1954) are timely.

They are timely because, although preliminary reports on serological prophylaxis are encouraging, epidemics must still be anticipated, and treatment, albeit symptomatic, has improved, even though the disease has appeared in severe form on a wide scale. Again, recent overseas epidemics seem to indicate a trend towards a higher incidence of bulbar and "scattered" paralysis, aggravated (as they might be expected to be here, too) by an influx of migrants more susceptible than the local populations.

I should like to comment on certain points in the articles referred to, in the light of experience in the 1952 Minnesota epidemic and elsewhere.

1. Muscular movement in the first two or three weeks. Hot-packing, followed by gentle stretching of "tight" muscle groups (precisely prescribed), was begun twenty-four to forty-eight hours after the temperature was normal, once to twice daily, avoiding fatigue. Reeducation (which here means assisted and attempted movement) was also begun at this stage and, by definition, concerns only the more paresed groups.

2. Hot-packing technique. Authorities should be aware of the value of the portable Vollrath Polio-Pak Heater. Munsing cloth (like "Jaeger" singlet material) was used, covered by a blanket, and over this, plastic cloth. Women orderlies "hot-packed" large numbers of patients readily by this means.

3. Muscle-charting and early assessment. Obviously, muscle-charting must not be done on a sick patient. But regular clinical observation of progression of paresis of muscle groups (including the diaphragm and intercostals, as noted by Dr. Robertson) is mandatory if close control of the patient is to be obtained. Muscle-charting, at any rate in some of the best American and European centres, is the responsibility of the physician, and not of the physiotherapist, who carries out the detailed treatment which the former prescribes on the basis of his muscle-charting.

4. Exercising. A consensus of reputable overseas opinion is to the effect that it is as important to prohibit indiscriminate efforts at muscle-strengthening in the early phase of physiotherapy as it is to ensure maximal progressive resistive exercise later.

5. Respirators. Two types of portable (cuirasse) respirator are commendable: the Technicon-Huxley Chest-Abdomen Respirator and the Monaghan Portable Respirator. The latter operates from accumulators and from mains. Emerson and Drinker-Collins tank respirators were used. They were very dependable. In common with most tank respirators, changing the position of the patient in them to prevent atelectasis could not be done as efficiently as desired; and much effort was needed in preventing contractures of shoulders, hips and heel cords.

6. Rocking beds. Emerson Rocking Beds were a godsend to the respiratory cripple, allowing him to receive daily physiotherapy (not to mention mere relief from the confining respirator); they also helped in weaning the patient from the respirator.

7. Positive-pressure breathing. (i) The Bennett Intermittent Positive-Pressure Breathing Unit (which fits the standard oxygen cylinder), although expensive, was very valuable, especially for preventing anoxia during nursing procedures on a severely affected respirator patient, and as a means of artificial respiration for respirator patients in a power failure emergency. (ii) The University of Minnesota Tracheotomy-Inhalator is a simple device (which can be readily made) permitting the supply of oxygen-enriched humidified mixtures to tracheotomized patients.

8. Education. It might be desirable to summarize the practical aspects of the essentials of the modern management of poliomyelitis in a concise illustrated booklet of the type issued by the United States National Foundation for Infantile Paralysis, for example, "Isolation Techniques and Nursing Care in Poliomyelitis" (which is, of course, a little out of date now).

Yours, etc.,

639 Sandgate Road,
Clayfield, N.2,
Brisbane.
January 20, 1954.

R. I. MEYERS.

COLLEGE OF GENERAL PRACTITIONERS.

SIR: Dr. D. W. Lawson, of Cessnock, Secretary of the proposed New South Wales Faculty of the above College, wrote an article in THE MEDICAL JOURNAL OF AUSTRALIA last year concerning the aims of the College and asking for practitioners who were interested to apply for forms of application for membership. I missed the article, as others may have, but joined because a very sound practitioner told me he considered it a sound idea.

Then the English *Practitioner* of December last came out with a long article on the history of the movement, its formation in 1952 and its proposed aims. In a nutshell, it envisaged the general improvement in the pre-graduate and post-graduate training of general practitioners and facilities for research work from their own point of view and within their own scope; all this to be directed by a council of their own members, who would from experience understand their problems. The movement is to embrace the British Empire and the United States of America.

It was stated that as the training of the undergraduate was solely in the hands of specialists, the special viewpoint of the general practitioner towards this work was not inculcated in that large and important body who for one reason or another did not desire to specialize after graduation.

Some men after graduation prefer to concentrate on one field of medicine only—portion because they feel possibly that their future will be easier if they keep abreast of one subject alone, and the rest because they are driven by a burning zeal to know every last word in their specialty and to excel therein. Their surroundings must be in the more congested areas of any country, so that they may be provided with the clinical opportunities which mass population provides.

Since only a relatively small percentage of people consult them privately, this same mass of population is necessary to provide their financial recompense. As the population spreads and thins from the large area they become scarcer and scarcer until they are non-existent. Others after graduation prefer the field of general practice. Some are content to go along quietly within their scope—accepting little responsibility and referring anything that worries them. Many others are attracted, however, by the great interest of the various fields of medicine and the close integration in the life of the community which it gives them. In contradistinction to the specialist, the general practitioner becomes increasingly important to the community as it thins and spreads, and he or she is their only present hope of safety.

This is the picture in this country rather than in the British Isles. On the whole their comparatively small size, closely settled population and highly developed system of transport keep them in touch with specialists of lesser or greater degree most of the time. Especially under the National Health Scheme is the general practitioner a sorter of sheep and goats without time to have a real clinical soul of his own.

The first proposal for this College came through a general desire to improve their training status and clinical opportunities. I am completely ignorant of the conditions obtaining in the United States of America.

We in Australia have greater scope—better standing and vastly better hospital clinical opportunities than our fellows in the old country. If this new proposal flourishes, our own

especial needs must be closely studied rather than that we should follow the lines laid down in the British Isles and Ireland—as our conditions differ greatly.

Since the best and latest information must be given to the student it is obvious that instruction should come firstly in each field from those who have specialized and excelled in their own sphere. Basic information must be of the very best available quality. Its method of application, however, is given only from the specialist point of view with its background of great hospital material and equipment. The young graduate who intends to become a general practitioner—unlike his fellow who specializes and carries on the rest of his life in the same sheltered atmosphere in which he was reared—when he leaves his training hospital may go to an area where clinical facilities are almost nil, and where his main assets are a lively imagination, powers of improvisation and courage.

Without claiming any of these virtues I can state that in my first small country practice my wooden splints were made by the undertaker and my metal by the blacksmith. I removed an appendix which was gangrenous by the light of one kerosene lamp in a small room, suspending the operation from time to time whilst the anæsthetist borrowed the light. On another occasion for lack of any help I "prepped" a "midder" and then confined her. She had an impacted breech, and I fractured a fœtal thigh whilst anæsthetizing her, delivering her and keeping her balanced on a small wooden table. I then washed the baby, dealt with its femur, cleaned up the mother and the bed, made her comfortable and gave us both a cup of tea.

Incidents like the above occur in the lives of general practitioners throughout the country each year. Young practitioners emerging from a specialist hospital are somewhat shattered when meeting crises of this nature. The occasional *placenta prævia* in wretched surroundings without trained assistance or any assistance can be devastating. And so, whilst the basic teaching of the various fields must be of the highest possible calibre, its application under the less specialized atmosphere of general practice, especially in the country, is not demonstrated. The new College proposes in some way to provide a practical outlook on the problems of its fellows based on impeccable teaching, and so standardize and improve their viewpoint. Many, left to their own caprice, court disaster.

An instance of how highly specialized things can become was shown to me in London a few years ago. I was watching a famous brain surgeon do a beautiful, intricate job on the mid-brain. All went well until just before the patient left the table. It was then noticed that the patient was cyanosed. There was a highly expert team in the room, and all looked stunned and did exactly nothing. This may seem incredible, but perhaps they were thinking only in terms of cerebral catastrophe. I somewhat diffidently suggested that perhaps if traction was applied to the tongue the patient might breathe. This actually proved to be the case, and the great surgeon glanced at me in disfavour and strode from the theatre after such a vulgar anticlimax to his high endeavour.

Group research in a new form into everyday problems will be organized if possible. Sir Norman Gregg's research in measles recently was "in the Jennerian fashion".

And so if general practitioners rally to their proposed College a more consolidated and generalized approach to their common problems may lie ahead. This very solidarity may be an enormous boon in time of crisis also. All are invited to investigate and join.

Yours, etc.,

T. HOLCOMBE.

Hornsby,
New South Wales,
January 22, 1954.

Post-Graduate Work.

INTERNATIONAL CHILDREN'S CENTRE, PARIS.

THE International Children's Centre, Château de Longchamp, Bois de Bologne, Paris (16e), founded in November, 1949, announces the following courses to be held in 1954:

The Influence of Prenatal Factors.

A course will be held dealing with hereditary and non-hereditary factors existing prior to conception, chiefly from the point of view of the practical application of existing information. The object is to aid medical practitioners in giving advice to families so that they may as far as possible avoid hereditary diseases. The course is designed primarily for pædiatricians, medical officers of health, administrators, statisticians and all interested in hereditary pathology. The course will therefore take the form chiefly of seminars and discussions. It will be held from March 8 to April 5, 1954, and 25 to 30 persons will take part.

Social Pædiatrics.

The course in social pædiatrics, which has already been held annually since 1948, will be held again in 1954. The programme covers all the important questions met with in medico-social and social work relating to normal or abnormal children. Its practical nature is emphasized by a number of visits to and periods of residence in medico-social centres and children's institutions, and particularly by the holding of a large number of discussions and study circles. The latter will be chiefly directed by those taking part with the aid of documentary material from their own countries. Team work among children's specialists will be particularly kept in mind. About 30 doctors from different countries are expected to attend the course, which will be held from April 26 to July 18, 1954.

Tuberculosis in Childhood.

A course will be held in the prevention and treatment of tuberculosis in childhood, designed primarily for pædiatricians and for health administrators responsible for tuberculosis control projects or for vaccination against tuberculosis, in connexion with the programmes of UNICEF and WHO. The instruction given will be presented chiefly in the form of demonstrations and practical work, supervised discussions and study circles; it will be based on experience acquired in a number of countries by various international vaccination teams. About 30 doctors will take part in the course, which will be held from October 11 to November 7, 1954.

Social Work for Children.

A course in social work for children will be given chiefly for social workers; it will be along the same general lines as the course in social pædiatrics. It will be mainly a practical course with a number of visits to and periods of residence in suitable institutions, designed to show the need for coordination in the work of social workers, doctors, social security organizations *et cetera*. There will be 25 to 30 participants, chosen from the highest ranking personnel among the social workers of the different countries. The course will begin on November 6 and continue till December 19, 1954.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 5, of January 28, 1954.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH

(SEA-GOING FORCES).

To be Surgeon Commander.—Surgeon Lieutenant-Commander Robert Michael Coplans (Acting Surgeon Commander).

AUSTRALIAN MILITARY FORCES.

Regular Army Special Reserve.

Royal Australian Army Medical Corps.

VX700337 Captain J. K. Francis relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 4th December, 1953.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The provisional appointments of the following officers are terminated, 14th November, 1953: Captains 1/39132 I. S. Holle, 1/39022 P. J. Landy and 1/61802 J. Brienl. To be Captains (provisionally), 15th November, 1953: 1/39022 Peter

James Landy, 1/61802 John Brienl and 1/39132 Ian Stanley Holle.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/146504 Colonel G. N. Young, E.D., from Assistant Director of Medical Services, Headquarters, 2nd Division, is appointed Deputy Director of Medical Services, Headquarters, Eastern Command, 1st December, 1953. 2/50455 Colonel A. E. McGuinness, M.C., relinquishes the appointment of Deputy Director of Medical Services, Headquarters, Eastern Command, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 1st December, 1953. The provisional ranks of the following officers are confirmed: Captains 2/127879 E. J. Hennessy and 2/127881 R. B. Geeves. The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain: 2/146562 F. J. Reynolds, 2nd April, 1951, and 6/15206 D. J. Walters, 14th November, 1953. The provisional appointment of F2/1075 Captain C. D. Chalmers is terminated, 14th November, 1953. To be Captain (provisionally), 15th November, 1953: F2/1075 Clair Drummond Chalmers, with regimental seniority next after 2/130107 Captain (provisionally) C. B. Saunders.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—To be Majors: Captains 3/101012 C. F. Macdonald, 4th December, 1953, and 4/31963 V. D. Plueckhahn, 9th December, 1953.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of 5/10586 Captain N. H. M. Colyer is terminated, 14th November, 1953. To be Captain (provisionally), 15th November, 1953: 5/10586 Noel Henry Maxwell Colyer.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

3rd Military District.—To be Honorary Captains, 2nd November, 1953: Alexander John Rollo and Ian Alexander Swain.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Flight Lieutenant J. R. Harrison (013663) is granted the acting rank of Squadron Leader, 7th September, 1953.

The resignation of Flight Lieutenant L. K. Rasmussen (025614) is accepted, 1st December, 1953.

Obituary.

GEORGE CRAIG HARPER.

DR. A. M. MCINTOSH has sent us the following appreciation of the late Dr. George Craig Harper.

Dr. George Craig Harper, who died at Burwood recently, was an exceptional character. Born in Melbourne in 1876, he had his early schooling at Toorak College, which his uncle, John Craig, had founded, and went thence to Ormond College within the University of Melbourne. He graduated M.B., B.S. in 1901 and, after spending a year in the Perth General Hospital, returned in 1902 to Sydney, where his father, the Reverend Andrew Harper, D.D., had been appointed Principal of Saint Andrew's College. For a year he was a resident medical officer in the Children's Hospital at Glebe. He then started practice in Temora, where, apart from his medical activities, he entered fully into the public life of the town. In 1916 he went abroad as a reinforcement to the Australian Imperial Force; he spent some time in the field, but served mainly with the First Australian General Hospital. On his return he established himself in practice in Burwood, where he spent the remainder of his life. He was devoted to his work and his patients, and in return he gained their affection and esteem. To most of them he was guide, philosopher and friend, sharing with them their sorrows and their tribulations, and doing much good by stealth when they were in need. He was the staunchest of friends, concealing under an appearance of austerity a very kindly nature, generous to a fault. He read widely, but with discrimination, and enjoyed more than

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 16, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1(1)	..	..	1	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	1(1)	9(8)	..	..	..	..	..	10
Diphtheria	3(2)	..	4(2)	..	2(1)	..	..	..	9
Dysentery (Bacillary)	..	..	1(1)	..	..	..	..	..	1
Encephalitis	1	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	4(2)	..	..	5(2)	..	..	..	9
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	1	..	..	..	1
Meningococcal Infection	1	1(1)	..	..	2	1	..	..	5
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	1(1)	..	..	..	..	1
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(6)	2(2)	1(1)	5(4)	5(5)	..	..	..	21
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	9(4)	1	..	23(18)	..	..	..	33
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	8(7)	13(8)	2(2)	2	..	..	..	..	25
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	..	..	..	..	1
Trachoma	..	..	..	..	3	..	..	..	3
Trichinosis	..	..	..	..	7(5)	..	..	..	7
Tuberculosis	36(28)	17(14)	12(10)	2(2)	..	6(1)	..	..	73
Typhoid Fever	..	..	..	1	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

anything else discussion with his friends. Of a rather shy, retiring disposition, he in general avoided public speaking, but he could be very fluent and forthright, particularly if he was roused by a sense of injustice. He wrote very little for publication, although he had a precise, cultured style best seen in his correspondence, to which he devoted meticulous care. Photography was his hobby and was a great solace to him after he retired from active practice—with this, as with everything he did, he took infinite pains and achieved excellent results. He was for many years a valued member of the boards of the Western Suburbs Hospital and the Presbyterian Ladies' College, Croydon, where his wise counsel and sound common sense were greatly appreciated.

George Harper's only son John was killed in New Guinea in 1945. He is survived by his daughter, Mrs. Lance Cunningham, of Melbourne. His sister, Dr. Margaret Harper, is well known in Sydney as a pædiatrician.

Congresses.

ANNUAL MEETING OF THE IRISH MEDICAL ASSOCIATION.

THE annual general meeting of the Irish Medical Association will be held at Killarney from July 7 to 10, 1954. Among those taking part will be Dr. Terence Millin, of All Saints Hospital, London, Dr. William O'Donovan, Director of the Dermatological Department, London Hospital, Sir James Spence, Professor of Child Health in the University of Durham, Dr. Ian Frazer, of Belfast, and Dr. T. J. D. Lane, Dr. P. MacCarvill, Dr. J. O'C. Donelan, Dr. W. R. F. Collis and Dr. J. Mowbray, all of Dublin. Visiting doctors and their wives and friends will be welcomed. In addition to the scientific meetings, an attractive social programme is planned.

Further information may be obtained from Dr. P. J. Delaney, Medical Secretary of the Irish Medical Association, 10 Fitzwilliam Place, Dublin. Information on hotels, tariffs and local tourist attractions can be had from the Irish Tourist Association, Upper O'Connell Street, Dublin.

Corrigendum.

IN the article by Dr. Keith Kirkland entitled "Tumours of the Adrenal Gland", an error was made in the acknowledgements. The name of Dr. Alison Garven should read Dr. John Garvan. We apologize to Dr. John Garvan and to Dr. Keith Kirkland for this error.

Deaths.

THE following deaths have been announced:

FELSTEAD.—James Gerald Roy Felstead, on January 26, 1954, at Melbourne.

CLENDINNEN.—Leslie Jack Clendinnen, on January 29, 1954, at Malvern, Victoria.

Medical Appointments.

Dr. A. J. Wood (nominated by Mount Isa Mines, Limited) and Dr. B. M. Andrea (nominated by the workers at Mount Isa Mines) have been appointed members of the Medical Board constituted in pursuance of the provisions of *The Workers' Compensation (Lead Poisoning, Mount Isa) Acts*, 1933 to 1945, of Queensland.

Dr. J. R. Mair has been appointed government medical officer at Mount Morgan, Queensland.

Dr. A. Maxwell-Allison has been appointed medical officer, Mental Hygiene Branch, Department of Health, Victoria.

Dr. C. S. Swan has been appointed honorary assistant ophthalmologist at the Royal Adelaide Hospital.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Grunwald, George Bela, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1950, 6 George Street, Manly, New South Wales.

Diary for the Month.

FEB. 13.—Tasmanian Branch, B.M.A.: Annual Meeting.
FEB. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
FEB. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 17.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 18.—Victorian Branch, B.M.A.: Executive of Branch Council.
FEB. 23.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.